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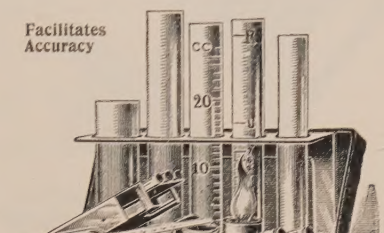

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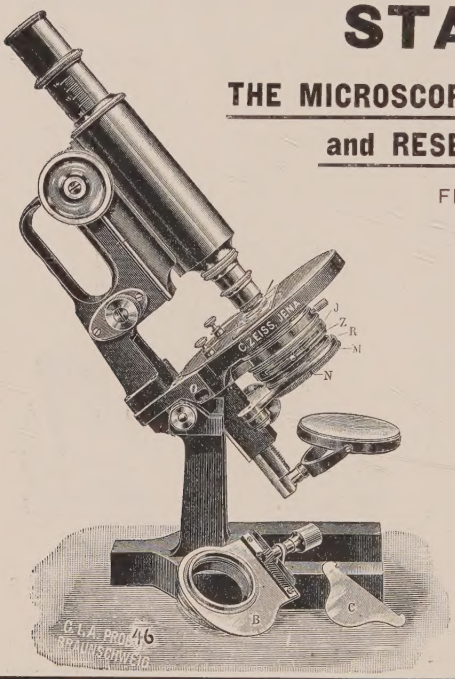

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TROPICAL DISEASES BUREAU.

TROPICAL DISEASES BULLETIN.

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PAPPATACI FEVER.

Annual Report of the Sanitary Commissioner with the Government of India for 1910.—1912. Calcutta: Superintendent, Government Printing, India. pp. 5-9 and 25.

Sandfly Fever in India in the Year 1910.—The term "sandfly fever" was introduced into the returns of statistics for the first time in the year 1910. The total number of cases recorded among the European and Native Troops is 1,058; they occurred in Peshawar, Nowshera, Attock, Kila Drosh, Chitral, and Kohat in the Punjab, and on the N.W. Frontier; in Bengal at Dinapore; in Rajputana at Neemuch and Mount Abu; in Bombay at Hyderabad; at the Himalayan Hill stations, Gharial and Dalhousie; and at Lucknow. This by no means represents the extensive prevalence of this disease throughout India. It is stated that it appears doubtful whether its great importance as a cause of sickness among the troops in India has yet been widely realised. Many cases were returned under the heading "pyrexia of uncertain origin" and several good clinical accounts of the malady appeared under that designation in the annual reports. There were, in all, 8,705 cases of "pyrexia of uncertain origin." The seasonal prevalence of 6,475 of these is given, and it is seen that 4,494 occurred during the months April to October, and hence probably were mostly sandfly fever. The *Phlebotomus papatasii* is found at Rawal Pindi; in the garrison of British troops, who numbered 2,861, there was a summer outbreak of 153 cases of fever which were registered "pyrexia of uncertain origin," though for the most part they were instances of sandfly fever. In many other stations similar errors were made. Again 257 of the 416 cases of influenza recorded in the statistics were admitted during the months April to October, and they occurred chiefly in places where the Phlebotomus abounds; they were therefore presumably sandfly fever, which has long been known as "summer influenza." We many conclude that during the hot weather of 1910 there were no less than 4,000-5,000 attacks of sandfly fever among the European and Native Troops of the Indian Army.

There is an excellent account of the symptoms of the fever and of the life history of the Phlebotomus. ANNANDALE has described

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six species found in India in addition to *P. papatasii*, but the habitat of the larvae has not been discovered. In 1908 PATEL obtained two larvae from drains, but there is no record of any finds in India since that date.

C. Birt.

Report on the Health of the Army for the Year 1911.—1912. Vol. 53.

The Incidence of Sandfly Fever among British Troops.—This report, which was published in December 1912, enables us to follow the incidence of sandfly fever in the European army of India during the year 1911. There were 1,393 admissions for this disease, which occurred at Peshawar, Nowshera, Attock, Rawal Pindi, Multan, Hyderabad, Kamptee, Quetta, Mhow, Lucknow, Meerut, and Lahore. Hence the true nature of these short febrile cases is becoming recognised more and more. "Pyrexia of uncertain origin" still accounts for 1,014 admissions, and "influenza" for 279, so that it is probable that many cases have been overlooked. The minute size of the Phlebotomus, its nocturnal habits, and its success in concealing itself during the day-time often cause one who is not an expert hunter to pronounce that it is not to be found, when it exists at a station. Since the bite of one sandfly may convey the infection, statements to the effect that short febriculas can not be sandfly fever, because no Phlebotomus has been discovered, must be received with caution.

At Malta there were 125 cases during the year 1911. At Cyprus one is recorded. In Egypt there were 96. At Tientsin and Peking 45 attacks were observed, which bore a general resemblance to the malady as seen in Malta, and occurred at the period when sandflies are most numerous. 54 of such cases are noted in the Hongkong returns.

C. B.

ROBINSON (S. C. B.) & BLACKHAM (R. J.). **Sand-Flies and Sand-Fly Fever on the North-West Frontier of India.**—*Jl. R. Army Med. Corps.* 1912. Oct. Vol. 19. No. 4. pp. 447-452.

The annual Reports of The Sanitary Commissioner with The Government of India from their commencement contain records of extensive yearly outbreaks of summer fever, now known to be sandfly fever, in Peshawar and other stations on the North-West Frontier of India. In the year 1911 this infection caused 848 admissions to hospital among the British troops of the Peshawar Division. The varieties of Phlebotomus that are found in the Peshawar Valley are *P. papatasii*, *P. minutus*, *P. molestus*, and *P. babu*. Sandflies are most numerous during the end of April and in May. Their numbers lessen in June and July; increase in August; diminish again in September and October. The flies disappear at the close of the latter month. The incidence of the fever fluctuates in like manner. Twelve officers made unsuccessful experiments to breed the insect from many varieties of material. They were seldom able to keep the sandfly alive in captivity longer than three or four days. Moisture favours their

survival. As preventive measures, the use of fine meshed nets, punkahs, and electric fans is recommended. It is stated that a brightly burning kerosene lamp appears to attract the flies more than the human being. Evacuation of barracks may be necessary. This was required at Kila Drosh. In 1910, 75 per cent. of the troops were attacked. The fort was evacuated on the first of June 1911, with the result that the incidence was reduced to 27 per cent.

C. B.

MANTEUFEL. **Notiz über ein bisher an der deutsch-ostafrikanischen Küste nicht bekanntes "Sommerfieber."** [Summer Fever in German East Africa.]—*Arch. f. Schiffs- u. Trop. Hyg.* 1912. Sept. Vol. 16. No. 18. pp. 619-622

During the hottest and driest months of the year in Daressalam a fever prevails which resembles pappataci fever in its duration and clinical features. Examinations of the blood and dejecta for malarial and enteric infections were negative. The epidemic subsides with the advent of the rains and cold weather. A Phlebotomus, the species of which was not determined, was found in the room occupied by one of the sufferers.

C. B.

MÜHLENS. **Malariaforschung in Jerusalem.** [Malaria Investigation in Jerusalem.]—*Deut. Med. Wochenschr.* 1912. Oct. 24. Vol. 38. No. 43. pp. 2036-2037.

During investigations concerning the prevalence of malaria in Jerusalem, the author learnt that an epidemic of fever had broken out at Jaffa. On proceeding there at the end of September 1912, he recognised the malady as pappataci fever. Phlebotomi are numerous both at Jaffa and Jerusalem every summer and cause much annoyance. He ascertained from the local practitioners that similar outbreaks occur annually in both cities.

C. B.

ERCOLANI (Aurelio). **Appunti Clinici sopra le Epidemie de Febbre dei Tre Giorni verificatesi a Forli nel Triennio 1909-1911.** [Three Day Fever at Forli, 1909-1911.] *Malaria e Malat. d. Paesi Caldi.* 1912. May-June. Vol. 3. No. 5-6. pp. 153-158.

Outbreaks of sandfly fever occurred among the troops at Forli (near Ravenna) in July and August of each year. The cases were much more numerous in some barracks than in others. The fever was not contagious. Its incubation period was under 8 days. The onset was sudden. The temperature rose rapidly to 40° or 40·5° C. There was severe frontal headache; pains in the back and lower limbs, especially in the knees; the conjunctivae were congested, and photophobia was often present. The tongue was furred, and sometimes vomiting and diarrhoea occurred. Mental depression was frequent and occasionally the patient became delirious. The

pyrexia declined gradually. In 80 per cent. of the attacks the pulse-rate was slow, often only 90 at the height of the fever, but might fall as low as 40. The leucocytes numbered 4,500-5,000 on the second day, falling to 3,000-3,500 on the third day. The mononuclears were relatively increased. The leucopenia continued till the eighth or tenth day. Bacteriological examination of the blood was negative. The duration of the pyrexia was usually 60 hours, but varied from 36 to 72 hours. Convalescence was slow, weakness continuing for ten or fifteen days. Acute nephritis was a complication in two instances.

C. B.

GABBI (U.). **Su di una Nuova Epidemia di Febbre dei Tre Giorni nella Sicilia Orientale. (Secondo ed ultimo contributo.)** [A Fresh Epidemic of Three Day Fever in Eastern Sicily.] *Studi Intorno alle Malattie Tropicali d. Italia Meridionale, d. Isole e d. Colonie.* Archivio trimestrale. 1912. No. 4.

An epidemic of pappataci fever broke out in Catania, Palermo and Partinico in the summer of 1911. The symptoms were milder than those which were observed in the outbreak of the summer of 1910; there were no deaths, nor severe complications; debility was less marked during convalescence than in the previous year.

C. B.

LEGER (M.) & SÉGUINAUD (J.). **Fièvre de Pappataci en Corse.**—*Bull. Soc. Path. Exot.* 1912. Nov. Vol. 5. No. 9. pp. 710-713.

There are annual summer outbreaks of a fever which resembles pappataci fever at Bastia and in its neighbourhood, in the northern part of the island of Corsica. It is very prevalent at Toga, a sea-side resort much frequented by the inhabitants of Bastia. The cases were less numerous in the summer of 1912 than in previous years, which was attributed to the greater frequency of the boisterous south-west winds. The *Phlebotomus papatasii* was captured at Toga. Seven temperature charts are given; in two cases the pyrexia lasted four days; in two, five days; and in the remaining three, seven, eight, and nine days respectively. The maximum temperature recorded in these is 105° F. Hence it is evident that the infection is of a more severe type than is usual, though it is a mistake to suppose that sandfly fever never extends beyond a period of three days.

C. B.

SEIDELIN (Harald). **Pappataci Fever.**—*Yellow Fever Bur. Bull.* 1912. July. Vol. 2. No. 1. pp. 74-84.

The bibliography of pappataci fever is given on pp. 116-120 of this number. More than one hundred references are quoted. Siedelin gives a concise summary of our knowledge on this subject.

C. B.

Phlebotomus and its Bionomics.

Langeron (Maurice). **Localités Nouvelles de Phlébotomes.**—*Compt. Rend. Soc. Biol.* 1912. June 21. Vol. 72. No. 22. pp. 973-974.

A. Weiss* reported the discovery of *Phlebotomus perniciosus* and *P. nigerrimus* on the island of Djerba off the Tunisian coast. Langeron agrees with the identification of the specimens. In Tunis he has met with *P. papatasii* only. The same species exists at Saint-Cyr au Mont-d'Or near Lyons. This locality is a connecting link between those in Bourgogne and in Valteline where its capture has been recorded. Saint-Vallier de Thiey (Alpes-Maritimes) and Montpellier (Hérault) are the only other places in France where it is known.

C. B.

Foley (H.) & Leduc (H.). **Phlébotomes dans le Sud-Oranais. Accidents Simplement Locaux dûs à leurs Piqûres.**—*Bull. Soc. Path. Exot.* 1912. July. Vol. 5. No. 7. pp. 511-513.

Phlebotomi are common in the oases of southern Algeria, particularly in the neighbourhood of Figuig. The species in that oasis, and probably also in the whole of the Sud-Oranais, is *P. papatasii.* The flies appear early in the hot weather, about the second fortnight in April, and disappear entirely at the end of the autumn in October or November. They become much reduced in numbers under the influence of cold and high winds, frequent inhabited houses only, where they hide in dark corners or behind curtains, etc., and avoid white-washed walls. The females bite during the day and night; newcomers often suffer severely. The irritation can be relieved often only by continual scratching; hence a pustular dermatitis frequently follows, which may be mistaken for scabies. Immunity against the bite is soon acquired by most people. The local inhabitants hardly perceive their attacks. No short fever is endemic in Sud-Oranais which might be attributed to these flies.

C. B.

Summers (Sophia L. M.). **A New Species of Phlebotomus from South America.**—*Bull. Entom. Research.* 1912. Aug. Vol. 3. No. 2. pp. 209-210.

Four specimens of a Phlebotomus were received from Rio Javary on the frontier between Brazil and Peru which were peculiar in the length of the head. This with the proboscis is, in the female, half that of the rest of the body. The wing venation resembles that of *P. malabaricus* (Annandale). In the male the greatest length of the wings is three and a half times the greatest breadth. They are bluntly pointed, and the hind border is not very much more strongly arched than the front. The length of

* See *Arch. Inst. Pasteur Tunis.* 1911. No. 4. p. 269, and 1912. No. 3. p. 199.

the 2nd marginal cell is contained $2\frac{2}{3}$ times in the length of the wing, and that of the third $1\frac{5}{6}$ times. The halteres are long and large. The name *P. rostrans* has been given to this new species.

C. B.

UNCLASSED FEVERS OF TROPICS, AND DENGUE.

Husband (James) & Hodge (H. V.). **A Peculiar Fever met with on the North-West Frontier.** [Mirror.]—*Indian Med. Gaz.* 1912. Aug. Vol. 47. No. 8. pp. 317-318.

The authors state that during the last two years they have noticed independently several cases of an obscure continued fever among the Sepoys on the North-West Frontier, in which they have failed to make a diagnosis. Eleven cases were observed having practically the same clinical picture; one of the noticeable characters was that according to the temperature chart the patient was suffering from a long and severe fever, though according to himself he was comparatively well. [This is a common characteristic of undulant fever cases.] All the cases were tested for typhoid and paratyphoid; one only gave a positive result at a 1/50 dilution for typhoid. Several were tested with the *M. melitensis*, but the results were negative, while no signs of tubercle or hepatitis which might have accounted for the fever were present. The authors consider that the cases were suggestive of short and mild infections of undulant fever, which is possible, as that disease undoubtedly occurs in the district; but in this fever there were no joint pains nor subsequent neuritis, and the fever did not last more than thirty days.

P. W. Bassett-Smith.

Taylor (D. M.). **The Relationship between "Pyrexia of Uncertain Origin" and Enteric Fever.**—*Indian Med. Gaz.* 1912. Oct. Vol. 47. No. 10. pp. 397-398. With 6 charts.

The author describes six cases of an infective disease which he had under treatment [place not stated]. The serum reactions were negative in all for typhoid, paratyphoid A. and B., and undulant fever; malarial parasites were not present. Three of the cases resembled typhoid clinically so closely that no other diagnosis was possible, two suggested paratyphoid, the sixth showed no definite character. An undoubted case of typhoid with a marked positive reaction died in the hospital at the commencement of the series. The author suggests not only that typhoid may occur in mild atypical forms, but that in these the reaction may be negative. This would account for many obscure fevers which are returned in India as pyrexia of uncertain origin.

[It would rather seem that we have to deal with an infective disease, not typhoid, but closely allied to it, of which the specific cause has not yet been determined.] The charts of the six cases are given.

P. W. B.-S.

Bradley (F. H.) & Smith (F.). **A Curious Case of Fever in Calcutta.**—*Jl. R. Army Med. Corps.* 1912. Aug. Vol. 19. No. 2. pp. 219-221.

This case occurred in a European soldier at the Jaffarpur camp, a few miles from Calcutta. When admitted to the hospital the patient was very ill, his conjunctivae were injected, and the face was dusky. A few days later a rash appeared which covered the face, extremities and body; this resembled the subcuticular mottling of measles, with small pink spots. The bowels were constipated. The patient's appearance became dull, the face more congested; urine was passed in the bed. There was then a sudden crisis followed by collapse, necessitating intravenous injections. After this he rallied and the fever did not return, but bed sores formed, and there was muttering delirium. He gradually improved, and six days after the crisis was out of danger. A diagnosis of typhus was made, but as there was considerable doubt, the case was returned as "Pyrexia of uncertain origin." Typhus occurs in the Punjab; it is possible that in some obscure way infection may have reached this man from the Pathans who come to Calcutta in the autumn. [The congested face, the character of the eruption, and the marked nervous symptoms are suggestive of a sporadic case of typhus, such as is occasionally seen at Shanghai. It is not stated whether there was a polymorphonuclear leucocytosis, which is generally a marked feature in the blood of typhus.]

P. W. B.-S.

Kennedy (R. S.). **Some Notes on an Epidemic of Dengue-Form Fever amongst Indian Troops, Calcutta.** [Mirror.]—*Indian Med. Gaz.* 1912. Nov. Vol. 47. No. 11. pp. 436-440. With 5 charts.

The author describes an epidemic of a fever of short duration which occurred during June, July, and August among the troops at Calcutta, and was at the time very common particularly in the northern part of the town. It chiefly affected the 40th Pathans at Alipore, and is stated to have spread with wonderful rapidity. Charts are given showing the curves of incidence of the disease, and the distribution of the cases throughout the lines. In July 319 cases were admitted to hospital, 194 of which came from the 40th Pathans, but all classes were affected, the disease passing from the sweepers' lines to the officers' quarters. No causative organism was found, but it was noticed that men admitted to the hospital for other diseases did not contract the fever, though they were sleeping in beds close to one another; this points to conveyance of infection from the sick to the healthy by some means which was not present in the hospital. After a review of the possible blood-sucking intermediaries, *Culex fatigans* was considered to be the most probable, as it was abundant both in the native lines and in the officers' quarters. An attempt to transmit the disease by means of mosquitoes failed owing to the death of the insects. A description of the clinical symptoms and many temperature charts are given. The former suggest dengue; the

latter show anything from a three day fever type to the typical double pyrexial attack common in dengue and seven day fever. The author believes that the epidemic was a form of dengue, but he forms no conclusion as to whether it was the same disease described by ROGERS as seven day fever.

[The description given by the author is exactly similar to epidemics of fever noted by the reviewer in the Defence flotilla force in Bombay, and then classed as dengue.]

P. W. B.-S.

CASTELLANI (Aldo.). i. **Cases of Fever probably due to** *Bacillus asiaticus.*—*Jl. Trop. Med. & Hyg.* 1912. June 1. Vol. 15. No. 11. pp. 162-166. With 2 charts.

ii. **Observations on some Intestinal Bacteria found in Man.**—*Centralbl. f. Bakt.* 1. Abt., Orig. 1912. July 17. Vol. 65. No. 4-5. pp. 262-269.

From two cases of obscure fever occurring in Ceylon the author isolated a peculiar bacillus which he calls *B. asiaticus* (1) and from two further cases a very similar organism termed *B. asiaticus* (2). Clinically the patients suffered from a low type of fever, two were infected with intestinal worms, and all had abdominal discomfort. In case 1. the organism was isolated from the faeces and the blood. A vaccine was prepared from it and, given therapeutically, appeared to cut short the fever. Specific agglutinins acting in dilutions up to 1 in 300 were present for six months in the patient's blood, but none were present for other common intestinal infective micro-organisms. In case 2. the organism was only isolated from the faeces, but agglutination reactions were present. Case 3. suffered from a low fever lasting four months, associated with abdominal pains. *B. asiaticus* (2) was isolated twice from the faeces. His serum gave negative reactions to all organisms except *B. asiaticus* (2) 1/80. *B. asiaticus* is described as a short, non-motile, gram negative, dysentery-like bacillus, which gives acidity with gas, in glucose, sorbite, saccharose media, etc., but gives no acidity or gas with adonite, lactose, or milk; the latter is often decolourised but there is no clotting. When freshly isolated it is pathogenic if given intra-peritoneally to guinea pigs. *B. asiaticus* (2) differs only in biological reactions.

The following is a summary of the author's conclusions.—In four obscure fever cases in Ceylon a new bacterium was found differing from any other intestinal bacterium. It occurs in two varieties *B. asiaticus* (1) & (2). The proofs of its being the causative agent are: specific agglutinins are produced, decreasing slowly in amount after the fever is over. The blood of the cases did not agglutinate other organisms, nor were other organisms isolated from it. The organism was only found in these cases.

A table is given of the cultural reactions of *B. asiaticus* and 76 other intestinal aerobic bacteria, which should be of considerable use for reference.

P. W. B.-S.

ii. In the course of the last eight years the author has examined the stools of a large number of persons in Ceylon, some healthy and others suffering from various diseases, and has found in the course of these examinations a number of bacteria not previously identified. He gives the names of thirteen species of these, of which two, *Bacillus asiaticus* (1), and *Bacillus asiaticus* (2), seem to be pathogenic, having been isolated from cases of fever and giving specific agglutinating reactions with the serum of the patients afterwards. Both these bacilli are gram negative, non-motile and typhoid-like in growth. The eleven other species were obtained from healthy individuals. The author gives an elaborate table of their sugar reactions, etc., in comparison with those of other intestinal bacilli.

S. R. Douglas.

Fox (A. C.). **Some Obscure Forms of Fever in North China.**—*Jl. R. Army Med. Corps.* 1912. Aug. Vol. 19. No. 2. pp. 209-211.

During the past three years, the author and other officers in the Tientsin district have noticed a peculiar fever among the European troops. Met with at all seasons of the year, it was most frequent during August, September, October, and November, that is, during and after the rainy season when flies were most troublesome. No malarial parasites were present in the blood and the fever was uninfluenced by quinine. The disease is locally known as "ten day fever," and has the following chief symptoms: Fever of a remittent type lasting ten to fourteen days, with frontal headache lasting two or three days; constipation, furred tongue, and slowish pulse. Some patients have abdominal discomfort and pains in the back. Constitutional symptoms were slight, relapses were rare, there was no mortality, and convalescence was rapid. Many of the soldiers who contracted the fever had quite recently been inoculated against typhoid. Major Fox thinks that this may be a paratyphoid infection, but he has not yet been able to have the serum tested.

[The sudden onset, regular course, and apparent protection produced by one attack certainly point to the disease being of a specific nature; the exact character will probably be worked out at Shanghai, where there is a good laboratory.]

P. W. B.-S.

Legendre. **Le Dengue, ses Variétés et la Conservation de son Virus en Indochine.**—*Bull. Soc. Méd. Chirurg. de l'Indochine.* 1912. Vol. 3. No. 7. pp. 456-462.

The author gives a list of cases of various degrees of severity, which he has observed. He comes to the conclusion that dengue has existed endemically at Hanoi since the epidemic of 1910, but in spite of the presence of so many non-immune persons it has not caused a severe epidemic. Most of the cases seen were ambulant or very mild ones, lasting from one to three days. These were not malaria, this disease being excluded by careful blood

examinations. The author shows that one attack of dengue is not always protective, and quotes the case of a child which had two attacks at an interval of 16 months.

His observations show, contrary to what has been noted by VASSAL, that the adult Annamites are susceptible to the virus of dengue.

P. W. B.-S.

POLECK. **Uber das Sechstagefieber in Apia.** [Six Day Fever in Apia.]—*Arch. f. Schiffs- u. Trop. Hyg.* 1912. Oct. Vol. 16. No. 20. p. 708.

The author, in continuation of previous work, makes observations on 12 fresh cases of this fever in the Somoan Islands. He notes that one attack does not give immunity and that leucopenia is well marked. He believes that the infection is conveyed by means of *Culex fatigans*, and that the causative organism is a long gram-negative streptococcus growing anaerobically.

[The details are few and no experimental proof is given.]

P. W. B.-S.

NÄGELE. **Über eine merkwürdige fieberhafte Erkrankung.** [On a Remarkable Febrile Disease.]—*Arch. f. Schiffs- u. Trop. Hyg.* 1912. Oct. Vol. 16. No. 20. pp. 705-707.

The author describes a peculiar febrile disease in German South West Africa. The symptoms are an urticarial eruption affecting both skin and mucous membranes, marked nervous disturbances, vertigo, loss of muscular power, neuritis, pain in the joints, and affection of the glands. The disappearance of the wheals is followed by exfoliation of the skin.

Relapses are frequent; the blood shows nothing distinctive. It is stated that the disease bears a resemblance to dengue fever during the first day, but differs in not being epidemic, in the frequent recurrence of the eruption, and in the comparative lowness of the temperature.

[The symptoms appear to point to some food toxin.]

P. W. B.-S.

DEEKS (W. E.). **A Preliminary Report on a Hitherto Unrecognized Six-Day Fever in Ancon, Canal Zone.**—*Jl. Amer. Med. Assoc.* 1912. Oct. 26. Vol. 59. No. 17. pp. 1511-1513.

PERRY (J. C.). **Outbreak of Seven-Day Fever in the Canal Zone.**—*U.S. Public Health Rep.* 1912. Nov. 1. Vol. 27. No. 44. pp. 1774-1775.

Deeks describes the occurrence of an infective fever of short duration affecting twenty persons all living in close proximity. Those admitted to the hospital were carefully studied. The disease appeared to be infectious in the early stage only, as it did not spread in the hospital though no special precautions were taken. The onset was abrupt and the temperature always fell to

normal on the sixth day, often after a precritical rise. The spleen was large; the kidneys showed signs of irritation; there was slight gastric discomfort. No marked change of pulse, respiration, or blood content was noticed, but general fever pains were present with an early erythematous rash, and in three cases a late scarlatiniform eruption.

The disease was at first thought to be malaria, but no parasites were ever found. From dengue it is distinguished according to the author by the large spleen, the single phase fever, and the absence of severe pains. He thinks the disease is identical with the seven day fever of India described by ROGERS, and that it was probably introduced with the mails from some infected port, as the first cases were Post Office workers.

Perry gives a description of the cases which occurred at Ancon with four additional ones admitted from a neighbouring village; he believes that the fever is identical with seven day fever as described by CASTELLANI from India and Ceylon, and states that it was neither dengue nor yellow fever.

[The fever resembles very closely the form of dengue described by ASHBURN and CRAIG in the Philippines.]

P. W. B.-S.

YELLOW FEVER.

SEIDELIN (Harald). i. **Report of Yellow Fever Expedition to Yucatan, 1911-1912.**—*Yellow Fever Bureau Bull.* 1912. Oct. Vol. 2. No. 2. pp. 123-242. With 3 plates and 2 maps.

ii. **Sull' Etiologia della Febbre Gialla.**—*Malaria e Malat. d. Paesi Caldi.* 1912. Sept.-Oct. Vol. 3. No. 9-10. pp. 245-246.

The outbreak of yellow fever investigated by the expedition lasted from August 3rd, 1911 to April 30th, 1912. The total number of cases officially diagnosed was 73, with 38 deaths. The disease was officially reported from Kambul, Maxcanú, Temax, and S. Felipe, and the author saw a case in Izamal. The experience of the last epidemic leads him to regard not only Mérida but the whole state of Yucatán as endemically infected. No importation of virus has been necessary to keep up the infection, but the arrival of a considerable number of non-immune foreigners has as a rule been sufficient to give rise to a severe outbreak with typical cases, whilst in the meantime cases with less typical and less severe symptoms have occurred in natives. The last outbreak was caused by the arrival of two battalions of soldiers from non-infected parts of the Mexican Republic. It is argued that yellow fever may occur with the severest manifestations of the disease in some cases while in others there is every transition to a milder type in which diagnosis may be very difficult or impossible. It is a recognised fact that in epidemics of yellow fever the new arrival is more liable to infection than the old resident and natives who have an acquired immunity. The author believes in the existence of a "*microbe carrier*" which is responsible for the maintenance of the virus during periods when the disease is latent and does not think that the fragile infected Stegomyia itself can be the lasting reservoir. Another point raised is the possibility of a second infection. The author considers that one attack does not necessarily confer an absolute immunity. He explains the usually mild form of the disease in natives as due either to an unrecognised infection in childhood or the heriditary transmission of antibodies from immune parents.

Thirty-five pages of the Report are devoted to a consideration of the cases observed and to the occurrence of yellow fever in natives. It is unnecessary to enter into a discussion of the clinical features of the cases. The author himself suffered from mild fever (maximum 100·4°) lasting two or three days and accompanied by slight jaundice, traces of albumin in the urine, and low pulse rate. Though five years before he suffered from what was considered to be yellow fever he again diagnoses his own case as one of this disease, in spite of the very mild nature of the attack. An examination of his blood failed to reveal any malarial parasites but on five occasions during ten days there was found the *Paraplasma flavigenum*, the bodies which the author considers to be the parasite of yellow fever and a further study of which was the main object of the Expedition. As regards the disease in the native the author has collected information from

the local medical men, many of whom are convinced that it occurs in them in a mild form. An account is given of some typical and severe cases occurring in natives who had been continuous inhabitants of the endemic areas.

Nineteen pages are devoted to the *Paraplasma flavigenum.* The author has seen his parasite in fifteen confirmed cases of yellow fever out of sixteen examined. The one negative result was due to the blood examination having been made too late in the disease. In one other case he found his organism in a blood film made at the commencement of a disease which was diagnosed as yellow fever post mortem. A table of the seventeen cases is appended showing the days on which the organisms were present in the peripheral blood. In two cases they were present on the first day of illness, in one case on the second day, in four cases on the fourth day, in five cases on the fifth day and sixth day. In one case they were found on the sixth, seventh, and eleventh to fourteenth days, while they were absent on the eighth and ninth and fifteenth days. The author sums up the results of his blood examinations for *Paraplasma flavigenum* in undoubted cases of yellow fever carried out on this and former occasions and finds that he has had approximately 106 positive cases out of a total of 120.

The author examined the blood of a large number of patients who were admitted to the lazaret on suspicion of yellow fever. They were non-immune foreigners with febrile disease. In thirty cases *Paraplasma* was found and of these thirteen were later diagnosed as yellow fever, while *Paraplasma* was found in seven cases seen in the city or country and of these three were officially diagnosed as yellow fever. Thus the parasite was found in 21 cases in which yellow fever was not diagnosed but which were all febrile cases of short duration with no definite local symptoms. Amongst the suspicious cases the author includes his own which was exceptional for a mild case in that the *Paraplasma* persisted in the blood even into the convalescent stage. In the mild cases the *Paraplasma* usually disappear quickly from the blood, so that they may have been overlooked in those admitted to the lazaret at a later stage of their illness.

In the section devoted to a consideration of the rôle and nature of *P. flavigenum* the author records the examination of 421 blood specimens as controls. In 136 cases malarial parasites were found and in 283 no parasites at all, while in two young children under no suspicion of yellow fever *P. flavigenum* occurred. The author recalls the fact that he recorded (1911) two similar cases which harboured *Paraplasma* and did not suffer from yellow fever. He suggests that these four cases may be "*microbe carriers.*"

As regards the morphology of the parasites themselves the author has nothing of essential importance to add to the descriptions which he has given in his earlier papers. No definite life cycle has been determined, but elements have been observed which "it can hardly be a mistake to consider" as division forms. The author adheres to his original view that the *Paraplasma* represents a new genus in the family *Babesiidae.* In answer

to criticisms by AGRAMONTE he tells us something of the number of *Paraplasma* encountered in his cases. He says that in his earlier series considerably more than three or four parasites had been observed in one smear though this was not the rule. It has not been the rule either in the last series but, exceptionally, larger numbers have been found. For instance in smears made from his own blood during his illness and in three from another case the author has "*repeatedly counted over forty parasites in a very thin film.*" [It perhaps ought to be remarked here that it is rather anomalous that the greatest number of parasites should have been found in the author's own case which he admits was of the mildest nature and which he includes only with the suspicious cases. The other case with large numbers of parasites was only seen late in the disease and the author had to rely on the patient's own account of the beginning of his illness which however conformed to the yellow fever view, since there was fever, haemorrhagic black vomit, melaena, jaundice and suppression of urine. When apparently recovering he suffered a recrudescence and it was then that he came under the author's notice in the lazaret. It would appear then that the number of parasites present in the peripheral blood bears no relation to the severity of the disease.]

Dark ground illumination was used but without very definite results. In one case, during the time that *Paraplasma flavigenum* was present in stained films, motile intracorpuscular bodies were seen which the author has no hesitation in identifying as his parasite.

Experimental work was conducted with guinea pigs. Blood from yellow fever cases was inoculated into four animals. None of them showed any symptoms resembling the disease. In two however structures, absent before, appeared in the red cells on the fourth and following days. These bore a striking resemblance to *P. flavigenum*, so much so that the author thinks that the organisms injected had multiplied to a certain extent in the animals and had invaded the erythrocytes but were unable to establish themselves to the extent necessary to produce any clinical symptoms. Experiments conducted with Stegomyia fed upon the cases gave unsatisfactory results.

In treatment salvarsan was given intravenously in one case in a dose of 0·1 gm. at noon and 0·2 gm. at 9 p.m. of the same day. No untoward symptoms followed the injections and the patient recovered.

The yellow fever section of the report closes with an account of a mosquito survey of Mérida followed by notes on the methods to be adopted for mosquito extermination, such as filling in and draining, petrolization and screening, fumigation with sulphur, etc.

In the final summary of his results the author writes:—

"*P. flavigenum* was found in practically all cases of yellow fever, in a number of suspicious cases, and in two apparently healthy native children; it was not found in other febrile diseases. I, therefore, consider it the pathogenic parasite of yellow fever."

Several pages of the Report are devoted to a reply to those who have criticised the author's work. SCHILLING-TORGAU* does not think the author's bodies are parasites but that they represent structures occasionally present within the erythrocytes of man and animals. AGRAMONTE† also objects to the view of the parasitic nature of the bodies, which he claims to have found in various conditions such as goitre, measles, mild biliary obstruction, severe icterus, uncinariasis, typhoid fever, and urticaria. He considers them to be "nuclear and protoplasmic fragments." CARTAYA and GUITERAS have also described intracorpuscular bodies from cases of glanders and icterus and they suppose them to be identical with the author's parasite. The author has examined specimens of the structures found by the last named workers and has no doubt that they are different from those in yellow fever. To the other critics he replies that the number of controls examined during the yellow fever epidemic in Yucatan is sufficient in itself to show that the structures which he describes as parasites are associated with yellow fever and absent in other conditions. He admits that "no observations have been published on the subject of this parasite" except his own.

Two coloured plates accompany the Report. Parasites are shown within red blood corpuscles and also in mono- and polymorphonuclear leucocytes. As seen in the coloured plates of this and previous publications the structures resemble some of the small piroplasmata such as *Theileria parva*. The author states (p. 178) that the forms usually observed were the small intracorpuscular bodies as formerly described. Free bodies were also found both in the peripheral blood and in smears and sections of the organs. [It must be an exceedingly difficult matter to recognise such minute structures in sections of organs and even when seen to be able to make up one's mind that they are identical with those seen in blood films. It must be evident to everyone reading this report that even if one accepts the statements that the bodies are present in cases of yellow fever and absent in other conditions the author has yet to prove that they are parasitic and if so are the etiological factor in the production of yellow fever].

C. M. Wenyon.

LE FANU (G. E. H.). **Medical and Sanitary Administration in the Protectorate of Togo.** Report to the Secretary of State for the Colonies. Received in Colonial Office, April 22, 1912.

The author gives a history of yellow fever in Togoland from the date of the first recorded case in Anecho in 1905. The disease almost certainly existed here before and was probably the

* SCHILLING-TORGAU (V.). Über die Bedeutung neuerer hämatologischer Befunde und Methoden für die Tropenkrankheiten. *Arch. f. Schiffs- u. Trop. Hyg.* 1912. Vol. 16 Beiheft 1. pp. 87–100: Zur Frage des Gelbfiebererregers. Antwort auf vorstehende Erwiderung des Herrn Prof. Dr. Seidelin. *Arch. f. Schiffs- u. Trop. Hyg.* 1912. June. Vol. 16. No. 11. pp. 373–376.

† AGRAMONTE (A.). Yellow Fever a Strictly Human Disease. *New York Med. Jl.* 1912. Sept. 7. pp. 465–468: Additional Note upon a so-called Parasite of Yellow Fever (Seidelin). *Med. Record.* 1912. Aug. 17. pp. 288–290.

cause of the transfer of the Government to Lome in 1895. From 1905 to the middle of 1911 there have been 27 recorded cases, of which 18 ended fatally. It is seen from the table of cases that in the years 1905 and 1906 there were 17 cases, only 4 of which were in natives. In the years 1907, 1910, and 1911 there were 10 cases, all in natives except one. No connection between the various outbreaks could be traced and the only conclusion possible is that the disease is endemic in the native population. It is generally held by the medical authorities in Togo that yellow fever, often in a slight and almost unrecognisable form, occurs among the natives, who are no more immune to yellow fever than they are to malaria. The places mentioned where infection occurred are Anecho, Grandpopo, Agoué, Lome, Badja, Tovega, Sebe, Anima, Misahöhe.

Clinical details and post-mortem findings of some of the cases are given and these present the typical picture of yellow fever infection.

C. M. W.

Colony of the Gambia. **Annual Report of the Medical Department for the Year 1911.** [Yellow Fever, pp. 33-38.]

Three outbreaks of yellow fever are reported by Drs. T. Hood and E. H. Read to have occurred in Bathurst during the year 1911. The first was in May with four cases and three deaths, the second in July with six cases and five deaths, and the third in November with a single fatal case. The characteristic symptoms, high death rate, and post-mortem findings leave no room for doubt as to the nature of the disease. The outbreak reported for 1911 bears a close similarity to the previous epidemic in 1900 in which there were eleven cases amongst Europeans and Syrians between May 23rd and October 30th. The death rate was about the same.

The five cases occurring in May 1911 were in Europeans, two of whom lived in a certain building over a soda-water factory, the other three being friends of theirs who were frequently in the same building. There is some evidence that a native Frontier soldier who was in charge of the soda-water machine was suffering from a mild form of yellow fever on May 8th. It is suggested that this man infected the Stegomyia which are always to be found in the dark ground-floor rooms. These mosquitoes probably found their way to the upper rooms where they infected the five Europeans, the first cases occurring on May 18th. Four other Europeans living in the same building did not contract the disease but this is accountable from the fact that they had not associated with the two inmates who fell sick. It is pointed out however that another explanation of the outbreak is possible. The S.S. Akassa from the coast was lying off the town on May 13th and 14th and at least four of those that had yellow fever went on board and remained there some hours. They may have been bitten by infected Stegomyia brought from some other port.

The second outbreak of the disease commenced on July 6th in a European who was living in a house next to others occupied by Syrians and natives.

The third outbreak consisted of a single fatal case in a European who was taken ill on November 10th. A definite diagnosis of yellow fever was only arrived at after the post mortem.

The active steps taken to check the spread of the disease consisted in the isolation of the cases in mosquito proof wards, the sealing up and fumigation by sulphur of the infected quarters and the establishment of four mosquito brigades to catch and kill mosquitoes in and about the European quarters. It is pointed out that so long as Europeans live in quarters close to natives outbreaks of yellow fever are liable to recur. To deal with Syrians and other non-immunes is more difficult, for they thrive in contact with the natives and short of prohibiting their presence in the town Dr. Hood is convinced that nothing much can be done to prevent their contracting insect-borne diseases.

C. M. W.

Augé (J.) & Pezet (O.). **Epidémie de Fièvre Jaune survenue au Dahomey pendant les Mois de Mai et Juin 1912.**—*Bull. Soc. Path. Exot.* 1912. Oct. Vol. 5. No. 8. pp. 648-656.

An account is given of an epidemic of yellow fever and the measures adopted to combat it in Dahomey during May and June 1912. The first case occurred in a young German 24 years of age. This proved fatal and was followed by a second fatal case which was probably infected from the first. On the occurrence of a third case enquiries were instituted in Bohicon, which was the infected centre, and it was found that previous to the first fatal case four Europeans had suffered from a form of fever which the authors consider to be the benign form of yellow fever that usually precedes the more serious cases. It was further elicited that the death rate amongst the natives had been unusually high, especially amongst the children and adolescents. Two travellers who had stayed in Bohicon were seriously ill on arriving at Cotonou. It was clearly shown that Bohicon was an infected centre and steps were taken to cut short the epidemic.—

1. Bohicon was isolated by forbidding departure by train except in the case of special officials and by the formation of a sanitary cordon to prevent movement of the native population.

2. The Europeans were persuaded to leave their residences and to live for a time in groups of not more than four in the neighbouring bush and not to communicate with one another or with any part of the town. Here they were watched for the development of any suspicious symptoms, in which case they were to be isolated in two rooms prepared for the purpose.

3. Steps were taken to destroy the mosquitoes already existing and to prevent the reproduction of any that should escape destruction. For mosquito destruction sulphur, tobacco, and formalin were used in the houses after closing all apertures, but it was found that the tropical house with its verandahs did not lend itself well to such a mode of disinfection. To prevent

mosquito reproduction, the usual method of filling and draining stagnant pools and the secure covering of wells and household cisterns was adopted.

Under this system of preventive measures the epidemic was stamped out, for only one case occurred after its institution and this must have been infected at an earlier date.

C. M. W.

Noc (F.). **Prophylaxie de la Fièvre Jaune et Travaux d'Assainissement de la Martinique.**—*Rev. d'Hyg. et de Police Sanitaire.* 1912. July 20. Vol. 34. No. 7. pp. 754-780.

This paper contains a detailed account of the measures adopted to check the spread of yellow fever in Martinique, where the disease is endemic and always appears in epidemic form when the Stegomyia becomes abundant. The island by reason of its geographical position is described as one of the chief sanitary barriers for the waters of Central America and the Panama Canal. The measures are those usually employed in combating the disease and are carried out under the direction of the Institute of Hygiene and Microbiology which is of official status and well supported by the government in its undertakings. Details of the staff employed in the administration are given and the various ordinances drawn up by the Government on the advice of the Institute are appended. The preventive measures consist in the early identification and screening of the cases, dealing with the breeding places of Stegomyia, the inspection of public lodging houses, boarding schools, and similar institutions where many individuals sleep under one roof, and education of the public to an appreciation of the reasons for the prophylactic measures. To prevent the transportation of the disease by ships calling at the ports, it is laid down that should cases occur on shore no person on board any ship is allowed to sleep on shore, or to be there at any other time than between 7 a.m. and 5 p.m.

It is pointed out that the measures employed against yellow fever are at the same time prophylactic against other diseases such as malaria and typhoid fever.

C. M. W.

Bertarelli (E.). **Risultati della Profilassi contro la Febbre Gialla dopo Dodici Anni di Applicazione.** [Results of Prophylaxis against Yellow Fever after Twelve Years' Experience.]—*Gazz. d. Ospedali e d. Cliniche.* 1912. July 23. Vol. 33. No. 88. pp. 913-914.

It is pointed out that in South America and elsewhere the prophylactic measures adopted against yellow fever have had most satisfactory results. The measures are roughly—

(1) Compulsory notification of even suspected cases; (2) isolation of these in mosquito proof wards; (3) immediate occlusion of all windows and other apertures in the house from which the case has come and the destruction of the imprisoned mosquitoes by

some suitable gas; (4) the prevention of pools of water, petrolisation, covering of water supplies, etc.

In Cuba in Havana city, in which the preventive measures were instituted in 1901, the deaths from yellow fever had been from 400-600 per annum. From 1901-1903 the death rate diminished very rapidly; in 1904 no deaths were recorded, in 1905 only one, and since then none. In Brazil prophylactic measures commenced in 1903 in the States of S. Paulo, Minas Geraes and the Federal District (Rio de Janeiro). In the town of Rio de Janeiro alone in some years there were as many as 2,000 deaths from disease; in 1905 a few deaths were recorded, but by 1906 there were none. In the northern parts of Brazil, where the preventive measures have not been satisfactorily instituted owing to the unhealthy climate, the scattered population, and other causes, the death rate from yellow fever is still high. Equally satisfactory results have followed the adoption of prophylactic measures in other countries.

C. M. W.

HARVEY (Christopher). **Epidemics of Yellow Fever at Bermuda.**—*Yellow Fever Bureau Bull.* 1912. July. Vol. 2. No. 1. pp. 13-73. With 1 map.

This is a paper of sixty pages which was written by the author in 1879, long before the discovery of the mode of spread of yellow fever. Consequently many of the conclusions are antiquated and erroneous. A detailed account of eleven epidemics is given. They occurred between the years 1699 and 1879 and the author endeavours to show that in every instance the disease was introduced to the colony *ab extra* by the arrival of a ship which had in some way been connected with epidemics of the disease in the West Indies and America.

C. M. W.

BLACKWATER FEVER.

FINK (Lawrence G.). **Blackwater Fever in Burma.**—*Jl. Trop Med. & Hyg.* 1912. March 15. Vol. 15. No. 6. pp. 81-88; and Oct. 1. No. 19. pp. 289-291.

In the first paper an account is given of five cases of blackwater fever. The geographical distribution of this disease, therefore, now includes Burma. The importance of ascertaining the distribution of malaria accurately and definitely in each province in India is pointed out. The author discusses the relationship between malaria and blackwater fever. According to DEADERICK, haemoglobinuric fever stands etiologically in the same relation to malaria as do tabes and dementia paralytica to syphilis, and so may very properly be regarded as a "paramalarial" infection. Malaria seems to be the essential cause of blackwater fever.

Blackwater fever has occurred in four districts in Upper Burma between 23° and 26° 30′ north latitude. Europeans and Indians, recently arrived, have chiefly been affected. As regards prophylaxis the author advises the reduction of cases of malaria by the use of quinine and gives a table showing the great decrease in malaria in a battalion of soldiers after prophylactic precautions.

In the second paper an account of nine cases of blackwater fever is given, and the author gives a summary of all the cases collected. It is evident from the figures that this disease occurs in several districts in Burma, and that Europeans, especially those on forest duty in very malarial districts and exposed to wet and chill, have suffered to a large extent.

J. G. Thomson.

JAMES (H. M.). **Comments upon Dr. Lawrence G. Fink's Article on Blackwater Fever, of September 15, 1911.**—*Jl. Trop. Med. & Hyg.* 1912. Oct. 1. Vol. 15. No. 19. p. 292.

The author points out that CRAIG's account of the geographical distribution of blackwater fever is decidedly erroneous; especially is he in error as to the prevalence of malaria and blackwater fever in India, and in Italy.

MARCHIAFAVA and BIGNAMI have defined blackwater fever as a syndrome that occurs in the course of a malarial infection, and the author thinks they are correct in this definition. He does not agree with these authors when they state that quinine cures some cases of the disease. We are unable, he writes, "to classify the result of quinine administration in blackwater, otherwise than to say that in some instances blackwater undoubtedly followed the use of the drug."

The author points out that parasites and pigment very rapidly disappear when once haemoglobinuria has set in. The value of early and abundant administration of quinine as a prophylactic against the development of haemoglobinuria in malarial cases was well shown in the report of ZEIGLER of the U.S. Navy. At present blackwater fever is seen in Ancon hospital in about one per cent. of the cases admitted for malaria. It occurs in those who have been severely affected by malaria and not in new comers.

J. G. T.

WOLDERT (A.). **Microscopic Findings in Twenty-four Cases of Malarial Hemoglobinuria.**—*New York Med. Jl.* 1912. Sept. 28. Vol. 96. No. 13. pp. 634-637.

Observations of a series of twenty-four cases of haemoglobinuria showed that at the onset of the paroxysm the urine, within a few hours, changed from the normal colour to a dark red, due to the presence of haemoglobin. This red colour did not depend on the presence of red blood corpuscles. None of a series of seventeen cases examined could be called haematuria. In eight of the seventeen cases red blood corpuscles were absent from the urine; in seven cases, and in two doubtful instances in which red blood corpuscles were found, they were never present in numbers sufficient to cause the portwine colour of the urine. No haemorrhages occurred from the kidneys. The reaction of the urine was in almost every instance acid; neither glucose nor indican was ever found, and no bile was detected.

There was a very large amount of albumin present, and also a great quantity of destroyed haemoglobin; in some instances the amount of albumin and blood colouring matter present after boiling amounted to 50 per cent. of the entire bulk of the urine. In some specimens of urine, bodies were found composed of uric acid; these were round and concentrically arranged, 10 to 40 microns in diameter, and often contained a dark brown dot or apparent nucleus. Bright red crystals of haematoidin were frequently found. Of the seventeen cases examined hyaline and granular casts occurred in seven, and were absent in ten. In nine of the seventeen, bodies like tube casts made up of disintegrated blood colouring matter, were found. No evidence was obtained to indicate that nephritis developed as a result of the haemoglobinuria.

J. G. T.

PICHOY. **Un Cas de Fièvre Bilieuse Hémoglobinurique observé à Diré-Daoua (Abyssinie).** [Clinique d'Outre-Mer.]—*Ann. d. Hyg. et Med. Colon.* 1912. July-Aug.-Sept. Vol. 15. No. 3. pp. 617-618.

It is generally believed, the author says, that blackwater fever does not exist in Abyssinia. As malarial fever is common in this region there seems no reason why cases of haemoglobinuria should not occur. A description of a typical case of blackwater fever is given. The patient had suffered from several attacks of malaria over a period of several years, and before the attack of haemoglobinuria had taken no quinine for several days. The urine was the colour of Malaga wine and spectroscopic examination showed the presence of haemoglobin. The patient was jaundiced and vomited bile-stained fluid. An injection of 250 cubic centimetres of Hayem's serum was given the day after the attack; four days later the temperature fell, the urine was free from haemoglobin, and the patient rapidly recovered.

J. G. T.

SKELTON (D. S.). **A Case of Blackwater Fever and a Suggestion.** *Jl. R. Army Med. Corps.* 1912. Oct. Vol. 19. No. 4. pp. 457-460.

The case described in this paper is remarkable in that no less than five years and eight months had elapsed since the patient left the West Coast of Africa, where he had a severe attack of the same disease. Except for a few doses of influenza mixture [? composition], he had taken no quinine for all this time; and in any case no quinine had been taken for four months. This, the author considers, rules out quinine as an invariable factor in the production of blackwater fever. The case was a typical mild attack of haemoglobinuria, such as paludics not uncommonly get on returning to England. The author thinks that in the case described here we have a malarial infection, the recrudescence of which can only be due to the parasite redeveloping by parthenogenesis.

He thinks it can be conceived that the large parthenogenetic forms of the *P. malariae*, containing as they do a large amount of nuclear matter and pigment, are so intensely toxic in their products as to give rise to the condition of blackwater.

J. G. T.

LEISHMAN (William). **The Etiology of Blackwater Fever.**—*Trans. Soc. Trop. Med. & Hyg.* 1912. Nov. Vol. 6. No. 1. pp. 1-20. With 1 plate.

Sir William Leishman first gives a review of the various hypotheses and observations on the etiology of blackwater fever, with a view to arriving at the points or factors which may be taken as established and upon which there is practically universal agreement. In the second part of his paper he describes the bodies encountered by himself in the leucocytes.

He draws attention to the acute differences of opinion on the etiology of blackwater fever. He groups all the hypotheses under one of the following four heads:—

(1) That blackwater fever is a manifestation of active malaria; (2) That it is quinine poisoning; (3) That it is due to a specific organism; (4) That it is a result of a condition brought about by previous malarial infection.

The first of these is little advocated to-day, and whatever influence malaria may have it is clear that it cannot alone be held responsible for haemoglobinuric fever.

Quinine poisoning as an essential cause is still a matter of debate. Sir Patrick Manson has recorded cases where attacks of haemoglobinuria could be produced at will by the ingestion of even a small dose of quinine. KETCHEN quotes a case where a series of seven attacks of blackwater were induced in the same individual; each attack was the sequel of a dose of quinine. The author quotes several strong points against its being due to quinine alone. The following are well established:—

"(i.) No cases have been recorded in non-malarious countries in which quinine, given for some other disease, has caused blackwater fever. (ii.) Numerous well-authenticated cases are on record in which blackwater

fever developed in individuals who had either never had quinine at all, or who had had none for a considerable time before the attack. (iii.) A man may have an attack of blackwater fever subsequent to a dose of quinine, and yet may afterwards take even large doses of the drug with impunity. (iv.) There appears to be no relation between the size of the dose, the channel of administration or the particular salt employed, and the attack, although each of these has at times been suggested as the essential factor."

As to the third view, namely that the disease is caused by a specific virus, there is at present no proof. SAMBON has suggested that there may be an organism analogous to a piroplasma.

The last view is most generally accepted, namely that blackwater fever, although not actually a manifestation of malaria, is a condition brought about by previous malarial infection. The excellent work of DEEKS and JAMES supports this hypothesis. The conclusions of these observers were as follows:—

"(1) Haemoglobinuric fever is a manifestation of malarial toxicity, for the most part brought about by repeated attacks of malaria. (2) It may appear coincidently with an acute malarial paroxysm. (3) It may be determined by any depressing influence. (4) It may be induced by the administration of quinine. (5) Quinine alone, or malarial infection alone, do not cause haemoglobinuria, but one or both of these conditions, plus the toxin eventuated during the course of one or more malarial attacks."

These authors also give the conditions most favourable for the production of haemoglobinuric fever; these are as follows:—

"(1) The presence of a population non-immune to malaria. (2) The prevalence of malaria in such quantity as to produce an almost continuous infection of this population. (3) A large proportion of aestivo-autumnal malaria; because the amount of blackwater fever is in direct proportion to the intensity of this variety. (4) The neglect of prompt and continuous administration of quinine, especially in primary attacks, to persons non-immune against malaria."

Various factors are given as the exciting causes of blackwater, namely, exposure, cold, damp, fatigue or over exertion, change of residence. Rarer exciting causes are mentioned. The most common exciting causes seem to be exposure to cold, and the exhibition of quinine.

CHRISTOPHERS and BENTLEY show that the haemolysin is probably derived as a result of auto-immunisation against the individual's own red cells. BARRETT and YORKE have shewn that quinine cannot be held responsible per se, since it would have to be present in the blood in a toxic dose before haemolysis could be produced; they believe that haemoglobinuria is the result of an antecedent haemoglobinaemia and that the anuria is due to mechanical blocking of the urinary tubes. CLELAND's recent theory is, that in the phenomenon of haemolysis we have an expression of an anaphylactic condition.

Sir William Leishman concludes the first part of his paper with the following summary of what might be recorded as well established points.

"1. Blackwater fever is only encountered in those who have either suffered from malaria or who have lived for some time in a country in which they must constantly have been exposed to the danger of malarial infection.

"2. One attack of blackwater fever confers no immunity against further attacks, but negro and other native races inhabiting endemic zones exhibit a certain degree of immunity. Whites and alien natives introduced into

an endemic zone are most liable to the disease, but, as a rule, only after a certain period of residence.

"3. Immunity to malaria appears to go hand in hand with immunity to blackwater fever, and measures which protect against malarial infection are also prophylactic against blackwater fever.

"4. The regions of most intense malarial infection are those in which blackwater fever occurs, but there exist also extremely malarious regions in which it is either extremely rare or absent.

"5. In the majority of cases an exciting cause is needed to produce the haemoglobinuric fever; the most frequent appear to be quinine and exposure to cold.

"6. The precise mechanism of the sudden and extensive haemolysis is not yet fully understood; there is certainly no direct relationship to the number or severity of the antecedent attacks of malaria, or to the number of malarial parasites present at the time of the attack."

In the second part of his paper the author gives a description of the cell inclusions found in the large mononuclears, which he thinks may be chlamydozoal in nature, and he also describes the "chrome cells" (see this *Bulletin*, No. 1, pp. 29-31). Sir William at first thought these were in some way connected with the inclusions, but since the publication of his first note he has received from D. Thomson of the Liverpool School of Tropical Medicine a communication in which the latter stated that he had observed similar cells in several different conditions, such as Hodgkin's disease, myelogenous leukaemia, carcinoma of the ileum, blood from leprosy nodules, and one or two others.

The author concludes with an examination of the conclusions reached in the first portion of his paper in the light of the chlamydozoal hypothesis.

(1) He suggests that the association of blackwater with malaria is comprehensible if it is assumed that the chlamydozoal virus is one which is transmitted either by mosquitoes or some different insect whose geographical distribution is similar. Mosquitoes transmit at least four diseases and of these yellow fever and dengue are due to filter-passing viruses.

(2) The second conclusion, referring to the immunity of natives in endemic areas, is not inconsistent with the new hypothesis if it is assumed that immunity follows the same lines in the two diseases.

(3) The conclusion that immunity to malaria appears to go hand in hand with blackwater fever supports the idea that the virus of the latter is also transmitted by a biting insect, similar in its habits to the mosquito.

(4) The fourth conclusion, which pointed out the discrepancies in the geographical distribution of the two diseases, and that blackwater fever only occurred in intensely malarious countries, is explained by assuming that some insect resembling the mosquito in its life habits transmits the specific virus; this insect must be either less numerous or not so widely distributed.

(5) The fifth conclusion that some exciting cause is needed, and lastly, (6) the conclusion relating to the mechanism of the production of haemolysis, both appear to await further understanding and explanation.

In conclusion the author suggests another possible connection between malaria and blackwater, namely the possibility of the malarial parasite itself being subject to disease.

J. G. T.

PROTOZOOLOGY.

MINCHIN (E. A.). **An Introduction to the Study of the Protozoa with Special Reference to the Parasitic Forms.** pp. xi. + 520. With 194 Text-figures and Bibliography. Demy 8vo. 1912. London: Edward Arnold. [21*s*. net.]

This text-book, written in Professor Minchin's well known facile style, marks an advance in Protozoology in England, as it is the first attempt by an Englishman to deal on a broad scale with the Protozoa.

The book can be divided into two parts. The first part, which consists of ten chapters, deals with the Protozoa from the biological and cytological point of view, and while of the greatest interest and charmingly written, it is probable that the second part (eight chapters), which gives a systematic review of the Protozoa, will appeal more to the worker in the tropics, who comes face to face with the organisms themselves. Hence this aspect is considered here somewhat more fully, though the first part will amply repay a careful perusal.

The nutrition of Protozoa, and especially of parasitic forms, is necessarily of economic importance. The organisms are considered under four types—(1) holozoic, (2) holophytic, (3) saprophytic or saprozoic, and (4) parasitic, and the inter-relation of host and parasite is discussed. The organisation of Protozoa, their structure, mode of life, provision for locomotion, excretion and sensitiveness are all described and illustrated. The physical and chemical constitution of the nucleus and nuclear bodies is discussed and interesting speculations as to the primitiveness or otherwise of chromation are dealt with at some length. The modes of reproduction of the Protozoa are detailed; this is highly important to workers concerned in the restriction of tropical diseases and in the elucidation of the cause of progress or relapse in individual maladies. The sex problem is one of the most speculative and controversial among some of the Protozoa, and a chapter is devoted to it. Professor Minchin states that while "sexuality is a universal attribute of all living beings above the rank of the Protista", it has not been observed with certainty among Bacteria and by no means among all the Protozoa. Syngamy (or sexual union) greatly increases the powers of recuperation possessed by the Protozoa. The necessity for syngamy can be greatly reduced by change of environment, and parthenogenesis and autogamy can replace it. The value of syngamy lies in keeping the tendency to variation within specific limits. Perhaps the subject of polymorphism is of greater practical importance to the worker in the tropics. Polymorphism may be due to either adaptation to environment, to growth and development, or to sex manifestation. Developmental forms of Protozoa also may be recapitulative as well as adaptive. The first part of the book closes with an interesting chapter on the general physiology of the Protozoa.

The second portion of the book consists of a systematic review. Two fundamental types of Protozoa are recognised: (1) The Sarcodine type and (2) the Mastigophoran type. The various classes are then considered in some detail.

The Sarcodina are treated under two sub-classes: (a) The Rhizopoda, comprising the Amoebaea, Foraminifera, Xenophyophora and Mycetozoa, and (b) Actinopoda, including the Heliozoa and Radiolaria. The group Amoebaea is of most interest to the tropical worker as it includes the pathogenic agents of amoebic dysentery. "The entozoic amoebae are commonly placed in a distinct genus, *Entamoeba*, distinguished from the free living forms by little, however, except their habitat and the general (but not invariable) absence of a contractile vacuole." The life-cycle of *E. coli*, illustrated by Casagrandi and Barbagallo's figures, is given in some detail and Prof. Minchin considers that *E. coli* "is not, under normal circumstances at least, a parasite in any sense of the word, but a simple scavenger, feeding on bacterial and other organisms, detritus, etc., in the colon and rectum." *E. histolytica* also is described, but illustrated only by Jürgens' figures—a pity when better and more modern ones are available. *E. tetragena*, which is of increasing importance, is not illustrated. This lack of illustrations will doubtless be remedied in a subsequent edition. The culturability of Amoebae is discussed at some length.

Among the Mastigophora, the sub-class Flagellata or Euflagellata is the most important. Their modes of life are discussed in great detail, and an interesting account of their methods of nutrition is given. *Lamblia intestinalis*, possibly a causal agent of diarrhoea, is well illustrated. The general classification of the Euflagellata is set forth.

The Haemoflagellates and allied forms, comprising Hartmann's so-called Binucleata, are considered under the generic types *Trypanosoma*, *Trypanoplasma*, *Crithidia*, *Leptomonas* (*Herpetomonas*), *Leishmania*, and *Prowazekia*. Twenty-six pages are devoted to Trypanosomes, and their structure, movements, life-history and effects on the hosts are discussed. The life-cycles of *T. lewisi* and of *Schizotrypanum cruzi* are described in detail and well illustrated. The life-history of *T. gambiense* is summarised so far as it was known in 1911. The typical life-cycle of a trypanosome in an invertebrate is considered to consist of three phases: (1) an initial multiplicative phase, (2) a crithidial phase, (3) a propagative, trypaniform phase. Prof. Minchin does not consider that the "granule" phase as described by Fry is a normal occurrence in the development of trypanosomes—a view accepted by the majority of workers on the subject. The genus *Crithidia* is shortly dealt with, but it is to be regretted that the statement on page 313 that, "up to the present . . . . no blood-sucking insect has been proved satisfactorily to harbour flagellate parasites not derived from vertebrate blood," is not in accord with the results of recent research.

The genera *Leishmania* and *Prowazekia* are discussed in some detail. Each chapter in the systematic portion closes with a statement of Prof. Minchin's ideas on the phylogenies and affinities of the various groups under consideration.

The Sporozoa are considered under two sub-classes, Telosporidia and Neosporidia. The Gregarines and Coccidia are well described and illustrated. The life-cycle of *Coccidium schubergi* is given and that of the interesting *Selenococcidium* is figured.

Among the Haemosporidia five principal types are treated in detail: (1) Haemamoeba type, (2) Halteridium type, (3) Leucocytozoon type, (4) Haemogregarine type, and (5) Piroplasma type.

The Haemamoebae include all the malarial parasites and the Proteosoma of birds, which are discussed in detail. The life-cycle diagram of *Haemoproteus columbae* is given. *H. stepanowi* is the type of haemogregarine considered, and the remarkable (but unconfirmed) work of Miller on *Hepatozoon perniciosum* is summarised. The piroplasms are classified according to França's structural varieties, and their development in the invertebrate hosts is discussed.

The Neosporidia are divided into Cnidosporidia and Haplosporidia. The former include Myxosporidia, Actinomyxidia, Microsporidia, and Sarcosporidia. Spore formation in the Myxosporidia is well illustrated. *Nosema bombycis* of pébrine is fully treated, and a good modern account is given of the Sarcosporidia. A summary, particularly of use to workers in the tropics, is given of *Rhinosporidium*.

The Infusoria are grouped as (1) Ciliata and (2) Acinetaria or Suctoria. The free living forms are fully described and very well illustrated. An interesting discussion of the affinities of the Ciliata indicates that it is highly probable that they are descended from flagellate ancestors.

The concluding chapter deals with the affinities and classification of the Protozoa and includes a brief note on the Spirochaetes. It is to be regretted that these highly important and difficult organisms are not more adequately treated and better illustrated. The increasing importance of the Chlamydozoa is recognised by a good, but unillustrated account of those organisms. The book of 520 pages includes a bibliography of 30 pages and contains 194 text figures.

Such is a brief, and necessarily inadequate, review of a very interesting volume.

H. B. Fantham.

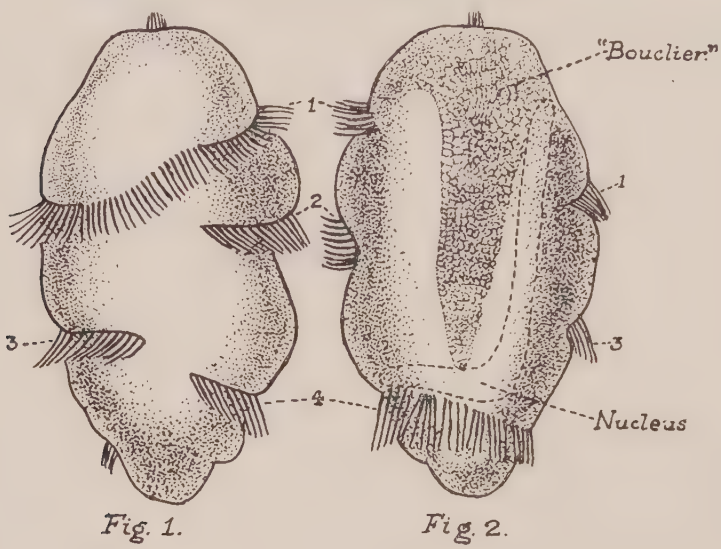
Brumpt (E.) & Joyeux (Ch.). **Sur un Infusoire Nouveau Parasite du Chimpanzé** *Troglodytella abrassarti*, **n.g. n.sp.**—*Bull. Soc. Path. Exot.* 1912. July. Vol. 5. No. 7. pp. 499-503. With 1 plate.

The authors found a new Infusorian belonging to the *Ophryoscolecidae* in the faeces of an adult female Congolese chimpanzee. Many parasites occurred in faeces neutral to litmus.

The average size of the Infusorian was 196·22μ by 115·11μ. Small ones measured 145μ by 87μ. The orientation of the parasite is difficult; the peristome determines the ventral surfaces. The body possesses four incomplete rows of thick, slightly motile cilia.

A digestive tube occurs with a lining of the same constitution as the external cuticle. The mouth possibly occurs at the junction

of the peristome with the pseudo-intestinal tube; in the latter starch grains have been observed, also faecal pellets at the cytopyge (cell-anus) which is situated at the summit of the 4th ciliated row. No contractile vacuole is present.



Troglodytella abrassarti Brumpt & Joyeux. Fig. 1 : ventral surface. Fig. 2 : dorsal surface. The numbers indicate the rows of cilia.

The macronucleus is shaped like an inverse S or L. There is no micronucleus. The endoplasm contains a cuticular supporting lamella, triangular in shape, and continuous with the shield (*bouclier*).

Asexual reproduction only is known. Division is transverse and fairly common.

The authors were unable to subinoculate the parasite into cats and monkeys. The parasite is rare in the chimpanzee and was found at autopsy to occur from the caecum to the anus.

The diagnosis of the genus *Troglodytella* given by the authors is:—

Ophryoscolecid, ovoid, posterior extremity not pointed, showing a thickened cuticular part (*bouclier*) on the dorsal surface prolonged into the cytoplasm as a sustentacular stalk. The body has four incomplete rows of large cilia. Type species *Troglodytella abrassarti*, Brumpt and Joyeux, July 1912.

H. B. F.

NAKANO (J.) **Wie verhalten sich die Kurloffzellen des Meerschweinchenblutes bei protozoischen Infektionen?** [The Behaviour of Kurloff's Bodies in Guinea-pigs' Blood in Protozoal Infection.]—*Folia Haematologica.* 1. Teil. Archiv. 1912. August. Vol. 14. No. 1. pp. 43-60.

The author commences with a historical résumé of the forms of leucocytes found in the blood and of the occurrence of Kurloff's

bodies. His own work was based on observations of three guinea-pigs, one of which was infected with *T. brucei*, two with *T. gambiense*. The animals were examined daily and detailed tables of the percentages of the various forms of leucocytes in the blood, the proportion of Kurloff's bodies present, the relative degree of infection of trypanosomes, and remarks thereon, are given. Curves demonstrating the daily variations of the different elements and of the Kurloff's bodies, are also given. A second series of curves shows the relation of Kurloff's bodies to the large lymphocytes.

The author's conclusions are as follows:—

(1) Neutrophile leucocytosis occurs in sleeping sickness in guinea-pigs and the number of lymphocyte cells undergoes reduction. This relation is reversed in nagana.

(2) In nagana of guinea-pigs the large lymphocytes are apparently the specific reactive or protective cells, while in sleeping sickness the polynuclear leucocytes are such.

(3) In various forms of experimental trypanosomiasis, whether lymphotactic or leucotactic, the Kurloff bodies gradually disappear from the blood and spleen, first increasing or diminishing in the blood in inverse ratio to the appearance of trypanosomes. No correlation exists between their decrease in the blood and increase in the spleen. The Kurloff bodies have apparently no direct connection with immunity processes.

(4) Kurloff's bodies show no morphological change resulting from protozoal injection of the host.

(5) Increase and decrease of Kurloff's bodies bear no relation to the increase and decrease of the azure granules in the small lymphocytes.

(6) Eosinophile leucocytes diminish in numbers in sleeping sickness of guinea-pigs; mast-cells increase to some extent in nagana.

H. B. F.

MARULLAZ (M.). **Sur une Hémogrégarine de** *Drymobius bifossatus* **(Raddi).**—*Compt. Rend. Soc. Biol.* 1912. Nov. 29. Vol. 73. No. 33. pp. 518-520.

The author considers that the organism serving as a basis for the description of *Drepanidium serpentium* (A. Lutz, 1901) was the haemogregarine of *Eunectes murinus*.

He examined the blood of two Brazilian serpents, *Drymobius bifossatus*, and found them parasitised by haemogregarines. He describes small, medium and large forms in the red blood cells. The larger forms are encysted therein. Free parasites are somewhat rare and are found more especially in the liver.

Multiplicative forms were found in the lungs and liver. They occur within oval cysts with very fine capsules. The contained merozoites are of two sizes, and are termed macromerozoites (2 to 10 in a cyst) and micromerozoites (16 to 24 in a cyst).

The author did not find "centrosomes" in the haemogregarines. He names the parasites *Haemogregarina drymobii*.

H. B. F.

HENRY (Herbert). **The "Infective Granule" as the Initial Phase in the Life-History of a Haemogregarine. (Preliminary Note.)** —*Brit. Med. Jl.* 1912. Dec. 14. pp. 1654-1655.

The contents of this paper, dealing with highly problematic stages of a haemogregarine (*H. simondi* in the blood of the sole, *Solea vulgaris*) are probably best summarised in the author's own words, thus:—

"While watching the escape of these parasites from infected corpuscles into the surrounding plasma I was struck by the fact that in many of the free haemogregarines one, two, or more bright refracting granules were found to emerge from the nucleus and to make their way through the protoplasm of the parasite to one or other extremity, in the majority of cases to the posterior extremity. In this passage from the nucleus to the posterior extremity these granules frequently divide . . ." "Their liberation into the surrounding blood plasma may be readily observed in films of fresh blood kept between slide and cover-slip, or in films made from blood kept in tubes of Nicolle's blood-agar medium" . . . "Red corpuscles are frequently found containing in their cytoplasm granules which are indistinguishable from and probably identical with the granules extruded from the haemogregarines" . . . "These intra-corpuscular bodies at first consist entirely of chromatin, but as they increase in size there appears round each a small clear zone of irregular outline, which zone later becomes a definitely stained piece of protoplasm. Phases showing this acquisition of protoplasm are met with occasionally in the blood stream, but more often in spleen and in liver smears. Also in spleen and in liver smears these bodies, composed of granules *plus* protoplasm, would appear to increase rapidly in size, so as to give rise to larger oval bodies which are distinctly haemogregarines."

The paper is illustrated by eight text figures.

[In the above paper, where the author compares granules of spirochaetes with those found in a piscine haemogregarine, it should be remembered that analogy is not homology].

H. B. F.

WOODCOCK (H. M.). **Notes on Sporozoa. Nos. ii, iii, & iv.** ii. Observations of *Karyolysus lacertae* (Danil.) and Remarks upon other Haemogregarines of Lizards. iii. Comparison of the Nuclear Condition in Haemogregarines with that in certain Coccidia. iv. The Nuclear Structure of *Leucocytozoon* and *Halteridium*.—*Quarterly Jl. Micros. Sci.* 1912. Sept. Vol. 58. Pt. 1. (New Series No. 229.) pp. 171-240. With 2 plates.

In the space at our disposal it is quite impossible to give a full summary of this long memoir. The protozoologist interested must consult it in the original. However, we would point out that:—

(1) In the first note the author states that there is "a remarkable agreement apparent between the nuclear condition of *Karyolysus* at a certain period of the life-cycle and that of a particular Coccidian in the corresponding phase."

The author "registers a strong protest against the habit of creating a new species on entirely insufficient grounds."

(2) In the second note, the author concludes that the Haemogregarine nucleus is not a distinct type but shows close agreement with the Coccidian nucleus.

(3) Lastly, in dealing with the nuclear structure of *Leucocytozoon* and *Halteridium*, the author discusses the significance of the so-called binucleate condition in these forms. He concludes that—

"In *Halteridium* the karyosome retains its central position within the nucleus throughout the period of growth of the gametocyte, and does not pass to the outside until the parasite is full-grown. On the other hand, in *Leucocytozoon* the karyosome appears to be always at the edge of, or else outside the nucleus." . . . . "In *Halteridium* the prominent extranuclear body is the karyosome of the nucleus."

"The association of *Halteridium* and *Leucocytozoon* (and also, in all probability, of *Proteosoma* and the malarial parasites) along with the Haemoflagellates in the group Binucleata has therefore to be given up. These Haemosporidia, equally with the Haemogregarines, must be regarded as closely allied to the Coccidia; it seems to me that there is no longer any reason for supposing that they are derived from a binucleate form, such as a Haemoflagellate. It has been a great disappointment to me to find that the view so elaborately worked out by Schaudinn and apparently so firmly based on facts, which I in common with many other Protozoologists adopted enthusiastically, has had to be abandoned, step by step, until the entire edifice is seen to be without any true foundation whatever."

There are figures of *Karyolysus lacertae*, *Leucocytozoon ziemanni*, *Halteridium noctuae* and *H. fringillae*.

H. B. F.

YAKIMOFF (W. L.). **Trypanosomes Parasites du Sang des Poissons Marins.**—*Arch. f. Protistenkunde.* 1912. Oct. 5. Vol. 27. No. 1. pp. 1-8.

The author gives a brief outline of the work on the trypanosomes of fish from 1841 to the present time.

He lists the fish examined by him at the Zoological Station of Naples during the winter of 1909-10. Two trypanosomes are described and figured:—(1) *Trypanosoma dorhni*, n. sp. in *Solea monochir* (one figure). (2) *Trypanosoma yakimovi* from *Syngnatus*. There are 8 figures of this parasite.

H. B. F.

SLEEPING SICKNESS.

Transmission.

Rodhain (J.), Pons (C.), Vandenbranden (J.), & Bequaert (J.). **Essais de Transmission du** *Trypanosoma gambiense* **par la** *Glossina morsitans.—Bull. Soc. Path. Exot.* 1912. Nov. Vol. 5. No. 9. pp. 762-770.

The experiments described in this note were made at Sankisia in Southern Katanga at a height of 750 metres (2,450 feet). Along the neighbouring watercourses *G. palpalis* and *G. morsitans* exist side by side, and sleeping sickness occurs. At Sankisia itself *G. morsitans* alone is found. *G. morsitans* bred from pupae were used. Eleven attempts at transmission were made with trypanosomes from six different patients; three were successful. Of these experiments four were made with flies which had fed directly on three natives with scanty trypanosomes in their blood; in others the flies were fed on an infected goat, a dog, or a guinea-pig; and in the three successful experiments on infected monkeys (*Cercopithecus*). The experiments are detailed. Of 81 flies used in the three successful experiments 63 lived at least thirty days and three became infective, that is to say, a proportion of 4·76 per cent. The details of the experiments do not differ from those given by other workers. In one case the infective fly was killed after 92 days; there was a typical infection in the salivary glands and three trypanosomes were found in the hypopharyngeal tube. In another experiment after the infective feed the flies were fed on a baboon for three weeks. In this case again trypanosomes were found in the salivary glands of the infective fly and a few in the hypopharyngeal tube. In the successful cases the trypanosomes were derived from two patients: in one instance the virus was of the Congo type; concerning the other the authors are uncertain.

Certain conclusions are formulated. It is noted that in the two infective flies examined there was no sign of multiplication of parasites in the proboscis; the trypanosomes found in the hypopharynx are believed to have come from the salivary glands. The latent periods in the flies were respectively 30, 35, and 24 days, which, it is noted, correspond with the times given by Bruce and by Taute, but are more than those found by Kinghorn and Yorke. It is noted that feeding on the blood of a refractory animal, the baboon, did not prevent the completion of the cycle of development. The authors write that in villages on the river Fungwe, near which *Glossina palpalis* is very rare or absent, the population has been decimated by trypanosomiasis; *G. morsitans*, which abounds there, may be playing an active part in the spread of the infection. The authors have not tried to infect monkeys with wild *G. morsitans*; their supply of laboratory animals is insufficient.

A. G. Bagshawe.

[illegible] *Notes on the Life-History of Trypanosoma gambiense, etc.* [illegible] Trans. Roy. Soc. [illegible] Plate 17, figs. 15. Vol. [illegible] No. [illegible] pp. [illegible]).

It is hard to say this paper is of the nature of a text-book and is not a full summary of the experiments and observations.

Endogenous Cycle in the Blood.—The author refers to the so-called Rosenbaum in the numbers of parasites present in the blood—the characteristic were made on particular and in the early stage in the type of infection. The crest forms, as he knows, upon an blood upon appears to be especially for certain day on the interval in the fly; the blood of a monkey is not influence by fly when blood forms and present in [illegible] and in a certain physiological condition, that is, age [illegible] form [illegible]. The differentiation into long and short forms is a phenomenon of growth and division and not an expression of sex.

Endogenous Cycle in the Fly.—Among the series of changes undergone in the fly up to the time when a sexual [illegible] or clear separation is very difficult and uncertain. The duration in time of this cycle varies in different [illegible] [illegible] [illegible] [illegible] [illegible]). The trypanosomes never appear [illegible] in the gut, they never disappear from the circulation. The gametes enter the body cells of the host and as they pass through the gut wall into the body cavity. The changes in the fly are described and figured. The first [illegible] in the gut are considered to be not very clear a [illegible] ideal phase in the young individual. This disappears before the appearance of the two gametes. The stages in the gut do not show any clear [illegible] phase. Incompletely [illegible] in the middle or posterior [illegible] and so [illegible]. It is noted that division is not longitudinal, but [illegible], and is [illegible]. [illegible] the fly [illegible] [illegible] [illegible] [illegible] [illegible]. This type in the gametocytes from and is the commencement of the development in the gut. Parasitic forms [illegible] [illegible] [illegible] [illegible]

almost indentical with the blood-forms but often a little below the normal adult length. These trypanosomes are found swimming free in the lumen of the glands and there is the strongest presumptive evidence for considering that these are the types that produce the infection in the vertebrate."

The development in the glands, the author writes, is the essential part in the whole cycle, the development in the gut being regarded as a mechanical device to enable the trypanosomes to establish themselves in sufficient numbers in contact with the salivary fluid, "which alone in the Glossina seems able to stimulate the trypanosomes to the apparently essential reversion to the crithidial type." Sexual differentiation has not been observed, but is not a characteristic of flagellate life-histories, isogamy being usual. It seems possible that the sexual part of the cycle might take place in the salivary glands. The author notes that there are no serious discrepancies between the cycle in the fly sketched by Bruce, Hamerton and Bateman and that described by her. She does not think the 'male' forms of Kleine and Taute play any important part.

A. G. B.

Kinghorn (Allan), Yorke (Warrington), & Lloyd (Ll.). **On the Development of** *Trypanosoma rhodesiense* **in** *Glossina morsitans.—Ann. Trop. Med. & Parasit.* 1912. Dec. 30. Vol. 6. No. 4. pp. 495-503.

The method of dissection of the flies has been previously described (see *Sleeping Sickness Bulletin* Vol 4. p. 235). The salivary glands attached to the pharynx are drawn out through the waist, so that the only lesion of the alimentary canal occurs in the anterior portion of the oesophagus; the danger of contamination from the intestine is thus reduced to a minimum. The authors found that in every fly capable of infecting animals with *T. rhodesiense* the salivary glands were invaded. 132 laboratory-bred *G. morsitans* used in transmission experiments were dissected as they died. Five were capable of infecting animals. In each there was an enormous invasion of the salivary glands by trypanosomes. In the 127 flies which remained incapable of transmitting the parasites the salivary glands were not involved, though trypanosomes were found in the intestine of 22. Data of the 27 flies in which trypanosomes were found are given in a table, showing the day of dissection after the infecting feed and the presence or absence of trypanosomes in the proboscis, intestine, and salivary glands. Trypanosomes were found in the proboscis in six instances between the 15th and 22nd days; in three of the infective flies they were not found (28th-40th days). The experiments with wild *G. morsitans* gave similar results. Of 620 flies dissected trypanosomes were found in the salivary glands of fourteen; all except four of these were proved to transmit the human trypanosome; in the case of the four the animals fed on died before a diagnosis could be made. None of the flies in which the salivary glands were not invaded was able to infect animals. Confirmatory evidence was obtained from the

G. morsitans caught in nature and fed on healthy monkeys. In all, twenty *G. morsitans* were found to have trypanosomes in their salivary glands and of these sixteen were definitely shown to be capable of infecting animals with *T. rhodesiense.*

The authors say that there was no doubt of the parasites being actually in the lumen of the tubes of the salivary glands. It was evident in fresh preparations, and in glands which were fixed after removal and subsequently embedded and cut the parasites could be seen inside the glands. In a fly which died on the twelfth day after being fed on a guinea-pig infected with *T. rhodesiense* a number of cysts containing trypanosomes were found in the mid gut; some had thin, others thicker walls; they ranged in diameter from 27 to 32μ.

It was found that on every occasion on which the salivary glands were infective the trypanosomes in the intestine were also virulent. This is strikingly shown in a table. In seven instances in which the salivary gland contents were introduced into animals and infected them the gut contents also were shown to be infective.

The authors write:—

"The manner in which the salivary glands become infected is uncertain, but there is a certain amount of evidence which would cause one to believe that it is secondary to the intestinal infection, and that it only occurs when the trypanosomes in the gut have reached a certain stage of development, and only then when the conditions of temperature are suitable for the further development of the parasites.....

"In none of our experiments were trypanosomes found in the salivary glands of flies which had not been subjected to the higher temperatures. Probably the salivary glands become invaded by parasites which have reached a certain stage in their developmental cycle in the intestine."

Invasion of the salivary glands was only observed in the case of flies infected with *T. rhodesiense* and not in the case of any other trypanosomes the authors met with either in the Luangwa valley or on the Congo-Zambesi watershed. As regards the trypanosomes found in the proboscis, they believe that their presence there is fortuitous, depending on the passage of the infected salivary secretion or on regurgitation from the gut, the result of manipulation during dissection. A description of the parasite as it appears in different portions of the fly is to be given in a further communication. The form in the salivary glands approximates rather closely to the short variety of the trypanosome in mammalian blood, but is not identical with this.

It is noted that of 510 wild *G. morsitans* dissected as they were brought to the laboratory mammalian red blood corpuscles were found in 70 and nucleated red blood corpuscles in four only.

[It will be noticed that Kinghorn and Yorke's account of the cycle of *T. rhodesiense* in *G. morsitans* agrees, as far as it goes, with that of Miss Robertson's for *T. gambiense* in *G. palpalis.* Miss Robertson however does not account for the infectivity of the intestinal forms, which was noted also by the Sleeping Sickness Commission in Uganda.]

A. G. B.

KLEINE (F. K.) & FISCHER (W.). **Schlafkrankheit und Tsetsefliegen.** [Sleeping Sickness and Tsetse Flies.]—*Zeitschr. f. Hyg. u. Infektionskrankheiten.* 1912. Dec. 20. Vol. 73. No. 2. pp. 253-259.

The authors recall that about four years ago KLEINE showed that *T. brucei* can complete its development in *Glossina palpalis.* It seemed then probable that *G. morsitans* might serve as a host for *T. gambiense.* Experiments were therefore undertaken on the shore of the Victoria Lake. TAUTE fed a large number of *Glossina morsitans* on sleeping sickness infected monkeys, but did not succeed in transmitting the disease to healthy monkeys. Of 120 flies dissected after 40 days trypanosomes were found in two; these were recognised as stages in the development, but not the terminal stage. It seemed that *T. gambiense* might commence its development in *G. morsitans,* but could not complete it (see *Sleeping Sickness Bulletin,* Vol. 1, p. 449).

Reference is then made to the transmission results of *BRUCE and his colleagues in Uganda, BOUFFARD in Senegal, BOUET and ROUBAUD in Dahomey, FEHLANDT in South Tanganyika, and FISCHER in North Tanganyika. It is clear from these that one species of Glossina may serve as host for various species of trypanosomes, and that the individual trypanosome species are in no way associated with individual species of Glossina.

It seemed probable that the negative result of the experiment on the Victoria Lake was occasioned by its height. This lake is about 1,200 metres above the sea, about 400 metres higher than Tanganyika. The Victoria Lake experiment was therefore repeated by TAUTE on Tanganyika. He used thirteen different human strains, two of which were brought in monkeys from the Victoria Lake. Of seventeen experiments fourteen were successful and it was shown in one experiment that four per cent. of the flies employed became infective (*S. S. Bulletin,* Vol.4, p. 269).

In order to see whether the negative result of the first experiment on the Victoria Lake was due to some undetected error or was the result of a natural law, the experiment was repeated in June, 1912, at the same place and at the same time of year. Six different strains of human trypanosomes were used and 1,402 laboratory-bred *Glossina morsitans.* Of fourteen experiments twelve were negative although they were continued for seventy days; two only were positive. One of these is given in detail. One hundred flies were fed for four days on a sleeping sickness monkey; between the 7th and the 25th day on three clean monkeys, which remained healthy; from the 26th to the 30th day on a monkey which sickened of trypanosomiasis; and between the 31st and 48th days on three other monkeys which likewise sickened. The remaining 47 flies were parcelled out and fed on various monkeys, and it was at length determined that a single fly was infective. In the other positive experiment two infective flies were found; that is to say, 0·21 per cent. of the flies employed

* In a footnote the opinion is expressed that what ZIEMANN described as *T. vivax* was a mixed infection of *T. cazalboui* and *T. congolense.*

became infective. Of 400 flies of the negative experiments which were killed after 70 days ten, that is to say, 2·5 per cent. had developmental forms of trypanosomes in their gut. The former result, therefore, was confirmed that the development of *T. gambiense* in *Glossina morsitans* may begin, but in spite of sufficient time is frequently not completed. It is clear, the authors write, that climatic conditions exercise a favourable or unfavourable influence on the development of the parasite in the flies.

They suggest that sleeping sickness is found chiefly in *Glossina palpalis* regions because this species lives in specially damp and hot places, that is to say, those which are favourable to the development of *T. gambiense*. It appears that *G. palpalis* is able to transmit the parasite at places where *morsitans* is not able. This, however, is not certain. Unfortunately no parallel experiments were made with *Glossina palpalis* and it is possible that at the place where *Glossina morsitans* could not transmit infection *G. palpalis* might at the same season have behaved similarly. They consider it possible that in sleeping sickness regions *T. gambiense* is not able at all seasons to complete its development in *palpalis*.

For the present their standpoint is that in Africa under suitable climatic conditions every known species of trypanosome—as *T. brucei*, *T. gambiense*, *T. congolense*, *T. cazalboui*, *T. nanum*—can develop in every species of Glossina.

[This paper should be read together with that of Kinghorn and Yorke, summarised in this *Bulletin* (No. 3, p. 126). Their work renders it highly probable that temperature is the essential factor, and humidity negligible.]

A. G. B.

Drug Prophylaxis and Treatment.

Duke (H. L.). **Some Experiments with Arsenphenylglycin and** *Trypanosoma gambiense* **in** *Glossina palpalis*.—*Proc. Roy. Soc.* 1912. Dec. 17. Series B. Vol. 86. No. B 584. pp. 19-31.

The experiments detailed in this paper were devised to investigate the action of arsenic upon *Trypanosoma gambiense* as carried by *Glossina palpalis*. Four separate enquiries were made.

I. Does the presence of arsenic in the blood imbibed clean an infected fly of its flagellates?—Boxes of laboratory-bred *G. palpalis* known to be infective were placed upon monkeys which had previously received a subcutaneous dose of arsenophenylglycin, 0·1 gm. per kilo. body weight. The mortality after the arsenic feeds was heavy. The flies were dissected at varying intervals. Seven such experiments were made, the interval between the administration of the dose and the feeding of the fly varying between 24 and 72 hours. Fourteen positive flies were obtained and dissected, and an account of the findings in each is given. It is seen that the flagellates in the gut were often much modified, with the posterior end swollen, and that many or all

were dead; sometimes none at all were seen in the gut. The forms in the salivary glands on the other hand were normal. It is noted that this difference had never been seen before in positive flies. The result of all the experiments is that the flagellates in the gut of flies fed upon a monkey within 24 to 48 hours of the administration of arsenophenylglycin in doses of 0·1 gm. per kilo. body weight are markedly affected. The flagellates in the salivary glands are apparently not injured in any way, nor does the fly lose its power to infect. The author remarks that this evidence supports the theory that the salivary gland flagellates are the normal infecting agents.

II. Has the preliminary feeding of flies on arsenic-containing blood any effect on the subsequent development of the flagellates in their interior?—Two experiments were made, one of which was positive. The flies were fed on an arsenophenylglycin-treated monkey and after a day's interval on a *gambiense*-infected monkey. Of these flies 2·6 per cent. were afterwards found infected with flagellates; of the flies fed first on a normal monkey (Control) 11·5 per cent. were found infected.

III. Does feeding on arsenic-containing blood, immediately after the infecting feed, prevent the subsequent development of flagellates in the fly, or does it result in the production of an arsenic-fast strain?—Two experiments were made. From them it appears that ingestion of such blood immediately after the infecting feed checks the subsequent development of the flagellates. The trypanosomes in a monkey which became infected showed no resistance to arsenic.

IV. Has arsenophenylglycin any prophylactic action against the bite of *G. palpalis* infected with *T. gambiense*?—Reference is made to a paper by Mesnil and Kerandel (summarised in *Sleeping Sickness Bulletin*, Vol. 3, p. 67), who tested the prophylactic action of the drug against inoculation of *T. gambiense* into monkeys. In one series of the present experiments 0·1 gm. per kilo. was inoculated subcutaneously; in the second series 0·05 gm. per kilo. It is noted that such doses applied to man mean a relatively enormous dose of arsenophenylglycin. Owing to the great sacrifice of experimental animals involved in testing the infectivity of each box reliance had sometimes to be placed on subsequent dissection of the flies to prove their infectivity. It is noted, as bearing on the experiments, that the mere introduction of the proboscis of a positive fly into the skin of its victim can produce infection (see *Sleeping Sickness Bulletin*, Vol. 4, p. 321), that a single positive fly can infect a clean monkey on three consecutive days, and that, as shown in (I), infectivity of the fly persists even after the gut has been cleared by arsenic feeding. The results are shown in two tables, one of which is reproduced.

It is concluded that the larger dose will protect a monkey against infection by positive *G. palpalis* if given twelve days before exposure (21 experiments) and that the small dose will protect if given within seven days (seven experiments). In Mesnil and Kerandel's experiments with doses of 0·05 gm. per kilo. three days was the limit of protection.

Experiment No.	Period between administration of arsenic and application of the + flies.	Number of experiments fed on monkey.		Number of + flies to which monkey was exposed.		Duration of examination of monkey.	Result.
		First proved infective on a clean monkey.	Not so tested.	1st day.	2nd day.		
	Hours.					Days.	
418...	24–48	1	...	1	1	52	—
438...	24–48	1	...	4	4	Died after 9 days.	?*
452...	24–48	1	...	1	1	45	—
487...	24–48	1	...	2	2	40	—
493...	24–48	1	...	2	2	41	—
524...	48–72	1	...	2	2	11	?†
620...	48–72	1	...	1	1	50	—
632...	48–72	1	...	2	2	60	—
654...	72–96	1	...	1	1	60	—
694...	72–96	2	...	3	3	62	—
661...	96–120	1	...	6	6	50	—
662...	96–120	1	...	2	2	42	—
692...	96–120	1	...	2	2	62	—
695...	96–120	1	...	2	2	63	—
689...	120–144	2	...	5	5	67	—
708...	144–168	3	...	12	12	61	—
711...	168–192	2	1	5	5	54	—
718...	192–216	6	1	12	6	60	—
694 (*a*)	216–240	2	...	3	3	55	—
782...	264–288	...	3	3	3	49	—
757...	312–336	...	2	4	4	...	+

* Died of arsenic poisoning after second dose of drug.
† Showed severe local reaction; killed.

The conclusions are as follows:—

"I. By feeding a *G. palpalis* which is infective with *T. gambiense* on an animal whose blood contains arsenic administered within 24 to 48 hours, the gut flagellates in the fly may be destroyed. Those of the salivary glands, however, which are the infecting forms, are unaffected. It is highly probable that the gut may become repopulated with flagellates from the salivary glands.

"II. Preliminary feeding of flies on arsenic-containing blood has a deterrent effect on the subsequent development of *T. gambiense* ingested within 24 to 48 hours after the arsenic blood.

"III. The feeding of flies on arsenic-containing blood immediately after the imbibition of *T. gambiense* usually prevents further devolopment of the trypanosomes in the fly. In the event of development occurring the strain produced is not arsenic-fast.

"IV. Arsenophenylglycin exerts a prophylactic effect in a monkey against infection with *T. gambiense* by positive *G. palpalis*; this effect varies with the dosage employed, and is considerably greater than when the trypanosomes are introduced by direct inoculation of infected blood."

A. G. B.

Daniels (C. W.). **Cases of Trypanosomiasis in England, mainly at the London School of Tropical Medicine. (Second Report.)**—*Jl. London School of Trop. Med.* 1912. Dec. Vol. 2. Part 1. pp. 91-95.

The author refers to his previous paper, published in the same journal, summarised in *Sleeping Sickness Bulletin*, Vol. 4, p. 18.

Seventeen cases were there recorded, of which five had died and twelve were alive. Eleven of these are known to be still living. Details may be seen in the table.

Case.	Initials.	Probable date of onset of symptoms.	Trypanosomes found.	Latest reports as to signs of disease.	General Result.	Duration of disease in years.
		Treated with Liq. arsenicalis and other drugs.				
1	H. K. (Ford and Dutton's case).	—	—	—	Death Jan. 1903, Cerebral symptoms.	$1\frac{2}{3}$
2	Mrs. M. ...	1900–1901.	July 1902.	None	Good health ...	11–12
3	Mrs. S. ...	1901	Oct. 1902.	—	Death Nov. 1903, Sleeping Sickness.	$2\frac{1}{4}$
4	J. M. ...	June 1902.	March 1903.	—	Death 1906, Sleeping Sickness.	4
5	Mrs. G. ...	?	1902	None	Good health ...	10 or more.
		Treated with Atoxyl.				
6	Z.	—	July 1905.	None	Good health ...	over 7
7	Mrs. R. ...	June 1905.	Sept. 1905.	None	Fair health ...	$7\frac{1}{3}$
8	H. C. C. S.	1905?	Feb. 1906.	None	Good health ...	about 7
9	W. R. E. ...	Sept. 1906.	Jan. 1907.	—	Death March 1908, Pneumonia.	$1\frac{1}{2}$
10	E. G. ...	Dec. 1907.	Jan. 1908.	None	Good health ...	5 or more?
		Treated with Atoxyl and Antimony either by mouth or by injection.				
11	C. G. ...	Nov. 1906.	Jan. 1907.	None	Good health ...	6
12	C. V. ...	July 1909.	June 1910.	None	Good health ...	$3\frac{1}{4}$
13	A. P. ...	July 1907.	July 1907.	None	Good health ...	$5\frac{1}{4}$
14	D. H. L. ...	Aug. 1908.	Aug. 1909.	—	Death 1911, Septicaemia.	$2\frac{10}{12}$
15	Dr. McD. ...	Dec. 1909.	Dec. 1909.	None	Good health ...	$2\frac{3}{4}$
16	W. G. ...	Sept. 1909.	Oct. 1909.	Present	Fair health* ...	$3\frac{1}{12}$ still infected.
17	C. W. B. ...	July 1910.	June 1911.	None	Good health ...	$2\frac{1}{4}$
18	H. R. T. ...	March 1912.	July 29 1912.	—	Died suddenly Aug. 26, 1912.	$\frac{5}{12}$
19	A. C. E. ...	April 1912.	May 1912.	—	Died suddenly Sept. 30, 1912.	$\frac{5}{12}$

* Died after an epileptiform seizure, Dec. 15, 1912 (Ed.).

The author considers that seven may be considered as recoveries and that of the other four three are probably on the way to recovery. The remaining case, W. G. (a Rhodesian case), was

described in some detail in the last paper. There has been little change in his condition; he still has attacks of pyrexia every eight to twelve days. Atoxyl is given regularly. The strain obtained from this patient is strain G of WENYON (see below p. 276). The two fresh cases described are also from Rhodesia.

H. R. T. was employed 300 miles north of Bulawayo and had never been north of the Zambesi. *Glossina morsitans* was common in his district and he did a good deal of shooting. His illness commenced in March 1912 with fever; he was admitted to the Albert Dock Hospital on July 31st. The features of his illness were the absence of all rash, late appearance of enlarged glands, testicular swelling, and sudden death on August 26th without pronounced nervous symptoms. He received atoxyl intramuscularly and rectal injections of tartar emetic. The strain from this case is strain T of WENYON.

The case of A. A. E. has been recorded by STANNUS (see *Sleeping Sickness Bulletin*, Vol. 4, p. 266). The symptoms commenced about April 4th, 1912; the patient was admitted to the Albert Dock Hospital on August 1st. In this case again there was no definite erythema; the glandular enlargement was slight; there was transient testicular enlargement; splenic enlargement, muscular tenderness, and anaemia were very marked. The patient left the hospital and died suddenly on September 30th. The strain from this patient is strain E of WENYON.

Commenting on these three cases, all due to the "variety (? species)" described as *T. rhodesiense*, the author says they are all of greater severity and more resistant to treatment than the cases seen from Uganda and West Africa. Orchitis was present in two of the three, whereas it occurred only in one of the other cases. Anaemia was very marked, as well as delayed muscular pain on trivial injuries. There were, however, no symptoms that have not been observed in patients from other parts of Africa. The trypanosomes in man were indistinguishable from *T. gambiense*.

The author discusses the position of the parasite and its relation to *T. gambiense*. With regard to the steady extension of trypanosomiasis south he thinks that expeditions have now done their work and that centripetal preventive measures must replace centrifugal observations of the spreading area of infection. An organised resident medical service will be required and one or more central laboratories.

A. G. B.

JOHNSON (J. T. C.). **A Proposed New Treatment for Human Trypanosomiasis.** [Correspondence.]—Lancet. 1912. Nov. 30. p. 1541.

Dr. Johnson calls attention to the fact that in 1895 Sir William ROBERTS read a paper on anarcotine, an alkaloid of opium, in the treatment of malaria. He regarded it as an antiperiodic of great power, analogous to, but not identical with quinine. The author suggests the use of this alkaloid in another protozoal disease, sleeping sickness. From his own experience he can say that it is

not harmful. He suggests that the drug be given in the form of pill, one, two, or three grains two or three times a day.

A. G. B.

T. RHODESIENSE.

WENYON (C. M.) & HANSCHELL (H. M.). **Notes on *Trypanosoma rhodesiense* from Three Cases of Human Trypanosomiasis.**—*Jl. London School of Trop. Med.* 1912. Dec. Vol. 2. Part 1. pp. 34-35.

This paper treats of strains G, E, and T from the Rhodesian cases described above by DANIELS. The trypanosomes in man never showed the posterior arrangement of the nucleus; these forms appeared first in the blood of rats inoculated from man. It is noted that though the posterior displacement occurs most commonly in the broad trypanosomes it is seen also in a small percentage of the very narrowest forms with long free flagellum. The percentage of posterior nuclear forms varies very much: in the G strain from 0 to 0·9, in the E strain from 3 to 7, and in the T strain from 13 to 40. In each case the percentages are based on counts of 1,000 trypanosomes made every three days. In other rats in the series the percentage remained constant except in the case of the E strain. Occasionally the G strain showed no posterior nuclear forms; in such cases it would be difficult to separate the trypanosomes from *T. gambiense*. The idea that some connection might exist between the virulence of the strain and the percentage of posterior nuclear forms proved unfounded. It is suggested that if a strain when inoculated from man into the rat contains many posterior nuclear forms the strain is likely to prove very virulent for the individual from whom it is derived.

A. G. B.

BIONOMICS OF TSETSE FLIES, AND CLEARING MEASURES.

LLOYD (Ll.). **Notes on *Glossina morsitans*, Westw., in the Luangwa Valley, Northern Rhodesia.**—*Bull. Entom. Research.* 1912. Nov. Vol. 3. No. 3. pp. 233-239.

The author, the Entomologist to the Luangwa Sleeping Sickness Commission, examined a small portion of the Luangwa Valley between August 1911 and March 1912. The physical features and climate are described. From June to December the whole valley is very dry; the vegetation consists of mopani trees with little or no undergrowth. The seasonal variations of *Glossina morsitans* are described; the species decreases in the dry season and increases in the rains. Notes are given of the habits of the flies in the valley and on the plateau. The author finds it advisable to have the bottoms of the trousers closed as the flies often enter and bite above the socks. With regard to the swarms of tsetse flies which accompany a moving object it is pointed out that the swarm consists almost entirely of males, just as is the case with midges; in each instance they are on the watch for females. Native fowls were used to feed the captive flies. The

blood was found to form large clots in the sucking stomach, which would persist for some weeks and prevented the retention of full-grown larvae. No evidence was obtained that the flies took any food other than blood. They did not bite Sphingid larvae when they had the opportunity. [ROUBAUD in West Africa found that *G. palpalis* would readily bite Sphingid larvae.] On one occasion the laboratory was raided by hordes of a red driver ant; these destroyed every fly, but did not hurt the pupae. A table is given showing the duration of the pupation period at various temperatures. Fifty-one days at 67° it gradually became less as the temperature increased till when this reached 85° it was twenty-three days. This is shown graphically in a diagram. The breeding of flies was much interfered with in the hot dry season, the number of pupae being reduced and a large number dying.

A. G. B.

NEAVE (S. A.). **Notes on the Blood-Sucking Insects of Eastern Tropical Africa.**—*Bull. Entom. Research.* 1912. Nov. Vol. 3. No. 3. pp. 275-323. With 2 plates.

In the course of his journey through Tropical East Africa, undertaken in 1910 and 1911, the author visited Nyasaland, British East Africa, Uganda, and portions of Northern Rhodesia and German East Africa. About 15,000 blood-sucking arthropods were collected. The following families are considered—Chironomidae, Culicidae, Simuliidae, Psychodidae, Tabanidae, Muscidae, and Hippoboscidae. The tsetse flies are dealt with on pages 299-309. Seven species are known from these regions.

Glossina morsitans.—An account is given of the answers to a series of questions on the habits of this species, issued by the Entomological Research Committee. Neave believes that among the essential factors which determine its distribution are a combination of the presence of such vegetation as will provide moderate but not excessive cover, and a hot and moderately or even very dry climate. He has often noted that when the grass is long (not less than four feet) *Glossina morsitans* is inclined to be more numerous in grassy areas of limited size than in the woodland or bush country surrounding them; he has seen this species swarming in long grass from half to one mile or more from the line of bush at the edge. Its complete independence of water is remarkable; the dryer the atmosphere the greater seems to be its activity. He considers that the presence or absence of big game is not the primary factor in determining the fly's distribution. He discusses the reason for the extension which is believed to have taken place in Northern Rhodesia and Nyasaland and expresses the opinion that the flies are recovering the ground lost at the time of the rinderpest. As to what caused this loss of ground he is uncertain, but it was not extermination of game because several species were little or not affected by the rinderpest. He thinks that *G. morsitans* can only be exterminated by extensive cultivation of the area it occupies. This must, however, be kept cultivated as it seems probable that not only *G. morsitans* but *G. pallidipes* will return to land where the bush has been allowed to grow up again.

G. pallidipes.—This somewhat resembles *G. morsitans* in its habits, but appears not to be so completely independent of water. It occurs mainly in the coast belt near the Equator.

G. austeni, Newst.—Neave caught this species in two localities in British East Africa. It is easily recognised by the bright rufous colour of the upper part of the abdomen. It is found in company with *G. pallidipes* and *G. brevipalpis* and would appear to be confined to the coast belt in British East Africa. It has not been captured higher than 1,500 feet above sea level.

The other species discussed are *G. palpalis*, *G. brevipalpis*, *G. longipennis*, and *G. fusca*. The notes are summarised in the following classification according to environment.—

"A. Requiring a great degree of atmospheric humidity:—
"A. 1. Requiring a high temperature. *G. palpalis*, R. D.
"A. 2. Not requiring a high temperature. *G. fusca*, Walk.
"B. Requiring only a moderate degree of humidity:—
"B. 1. Requiring comparatively little cover. *G. pallidipes*, Aust.
"B. 2. Requiring fairly heavy timber and bush. *G. brevipalpis*, Newst.
"B. 3 Requiring more or less dense forest. *G. austeni*, Newst.
"C. Independent of water and most active in a dry atmosphere. *G. morsitans*, Westw., and *G. longipennis*, Corti.

"The three large species, *G. brevipalpis*, *G. longipennis* and *G. fusca*, may also be separated from the others by their being mainly crepuscular or nocturnal in their habits, instead of being most active in sunshine."

At the end of the paper lists are given of blood-sucking arthropods arranged according to locality. In a note it is suggested that the *G. tachinoides* recorded from German East Africa may prove to be *G. austeni*, Newst. The paper contains two plates, illustrating twenty *Tabanidae*.

A. G. B.

Fell (T. E.). **Notes on Tsetse-Flies and on Prophylactic Measures against Sleeping Sickness in the Western Province of Ashanti.**—*Bull. Entom. Research.* 1912. Nov. Vol. 3. No. 3. pp. 227-231.

Mr. Fell, Provisional Commissioner, Ashanti, gives data of the habitat of the tsetse flies in this district, namely, *G. fusca*, *G. palpalis*, *G. pallicera*, and *G. longipalpis*. The common species are *palpalis* and *longipalpis*. Tsetse-flies practically disappear from December to the end of March and reappear when the rains are thoroughly established. An effective clearing was made at Sunyani and is maintained by the constant labour of thirty to fifty men a day, but odd flies still appear in the European quarters. The author has convinced himself that a forest clearing not effectively maintained produces a scrub growth which is more favourable to tsetse flies than the forest itself. Thus a position of affairs may be produced which is more dangerous to the population than that which has hitherto existed. He doubts whether the small populations of the villages can effectively maintain the clearings which are at present advocated. As to clearings round village water supplies he points out that the removal of shade may diminish the water, if not dry it up altogether, causing a water famine. He does not think that roads at river crossings can be effectively cleared unless by Government labour at great expense, which in his opinion is at present

unwarranted. Moreover, in the present state of sleeping sickness in Ashanti the object of enforcing clearings would not be understood by the natives and compulsion would tend to make them conceal cases. He points out that where the disease is most prevalent *longipalpis* exists together with *palpalis;* he suggests that that species may be a transmitter. In conclusion he urges—

"1. That the native should be encouraged to report cases of sleeping sickness for diagnosis and treatment, and to give evidence, and should not be frightened by excessive compulsory work into concealment of the disease.

"2. That the population is not sufficiently large to maintain extensive effective clearings.

"3. That a non-effective clearing of forest produces a condition probably more dangerous than no clearing at all.

"4. That clearing round water supplies should only be undertaken with the greatest caution.

"5. That clearings at river crossings in forest are impracticable unless undertaken with Government-paid labour, and that the cost of clearing and maintenance is not at present warranted, owing to the limited occurrence of sleeping sickness"

At present he would advocate object lessons of extensive clearings round European stations and the upkeep of village clearings of such an extent as can be effectively maintained by the population, the extent to be left to the discretion of the administrative officers. Finally, he gives notes about the wild mammals of this district.

A. G. B.

Trypanosomiasis in Nyasaland and Belgian Congo.

In Part 18 of the *Nyasaland Sleeping Sickness Diary,* dated September 30th, 1912, by H. Hearsey, P.M.O., seventeen additional cases of sleeping sickness in natives, diagnosed by Dr. Shircore, are described. These with the 76 previously recorded make a total of 93. Five of the patients died shortly after trypanosomes were detected; details are given of twelve, ten men and two women. In eleven of these tremors of the hands, and more rarely of the tongue, are noted. In ten there was oedema of some part of the body. In most the gait is described as shuffling. The glands of the posterior triangle are described as small, not puncturable, not palpable, or at most a small chain, in eleven of the cases; in the twelfth they were enlarged and puncturable. In many the epitrochlears seem to have been enlarged. In two cases, in adult males, interstitial keratitis is recorded.

It is noted that the medical officers investigating in the districts adjacent to the sleeping sickness area had not up to the present found any cases; this failure may be attributable to concealment of the sick. Some notes are given of the use of a dye in treatment.

[Interstitial keratitis, common enough in trypanosome infection of animals, has been recorded only once before as a symptom of human trypanosomiasis, by Thiroux and d'Anfreville in 1911.*]

A. G. B.

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* *La Maladie du Sommeil et les Trypanosomiases animales au Sénégal.* A. Thiroux & L. d'Anfreville. 1911. Paris : Librairie J.-B. Baillière et Fils. p. 23.

In the Report on the Administration of the Belgian Congo for the year 1911, under the heading Medical Service, it is stated that there are 27 lazarets for sleeping sickness patients. These establishments are intended chiefly for natives in the last stage of the disease; others stay only till the diagnosis is confirmed. The number of patients treated in lazarets for the financial year 1911-12 was about 4,000. Ambulatory medical treatment is very generally employed, by doctors who travel in the neighbourhood of their stations. An enormous number of patients have been thus treated; at Beni, for instance, 1,000 patients per week. It is noted that this form of treatment can be undertaken by laymen. There were 34 clearing brigades with an effective strength of 1,700 men. Owing to the wide distribution of *G. palpalis* removal of villages has been practicable in hilly districts only. As a rule small villages are collected into larger, which are better able to keep up proper clearings. It is noted that the disease has been particularly severe in the basins of the Semliki, of Lake Kivu, of the Kwilu, and of the Kwango rivers. The sum of 155,000 fr., obtained by the sale of flowers and put at the disposal of the Administration by the Queen, will go to the erection at Leopoldville of a model lazaret, the training of 20 hospital assistants, and the purchase of instruments and medicines for the combating of sleeping sickness; these will be entrusted to missionaries trained to use them.

A. G. B.

Unclassed.

Lafont (A.). **Trypanosomide d'un Réduvide** (*Conorhinus rubrofasciatus*) **Inoculable au Rat et à la Souris.**—*Ann. Inst. Pasteur.* 1912. Nov. 25. Vol. 26. No. 11. pp. 893-922. With 2 plates.

Conorhinus rubrofasciatus is a Reduviid bug known in Mauritius as "*Punaise maupin.*" Specimens from Réunion were also studied. The biology of the Hemipteron is set forth in detail in the latter portion of the paper. The insect is a blood sucker, which will attack man.

The flagellates commonly occurring in the digestive tract of the bug are described. They are found chiefly in the intestine, rarely in the stomach, and never in the salivary glands, Malpighian tubules, ovaries and haemocoel. The flagellates were studied in life and in stained preparations.

The author describes in detail (1) various crithidial forms (short, medium and long); (2) small trypanosome forms, with large blepharoplast; (3) rounded forms, some of them like *Leishmania*; (4) cystic forms, elongate or fusiform; (5) dividing and abnormal forms. The morphology of all these is set forth in detail with figures.

Attempts at culture were not very successful, the parasites being merely conserved for a few days.

The author tried to transmit the flagellates of the *Conorhinus* intestine to mammals. Attempts by insect bites on *Macacus cynomolgus* and guinea-pigs failed. Subcutaneous inoculation

of intestinal contents of the insects in Macacus, rats, guinea-pigs, rabbits, etc., also failed. Intraperitoneal inoculation of intestinal contents succeeded only in the case of rats and mice. Living flagellates were seen in the peritoneal fluid of the inoculated rats up to 30 hours, encystment and phagocytosis occurring. These rats were free from *T. lewisi*, but all died from various undetermined causes. In inoculated mice of Mauritius the trypanosome seen in the blood was three to four times the size of the small trypanosomes seen in the Reduviid intestine. In inoculated mice of Cilaos the trypanosome was smaller than in Mauritius mice. In the mouse the trypanosome has a blepharoplast which is near the nucleus, therein differing from *T. lewisi* and *T. duttoni*. Dr. Lafont also shows that the parasite is not *T. evansi*.

The author discusses his results more particularly from the point of view of whether the Reduviid harbours a trypanosome of another animal. The flagellate in the insect is polymorphic. Intermediate forms leading to the larger trypanosome were seen. The name *T. boylei* has been given to the trypanosome phase in the rodent's blood, with its developmental phases in *Conorhinus*.

Perhaps *T. boylei* may be pathogenic to man, and transmitted by *Conorhinus rubrofasciatus*. The author hopes that his data will lead some worker, more fortunate than himself, to discover a new human trypanosomiasis.

[In the space available it is, unfortunately, not possible to give a detailed summary of this interesting and well-illustrated memoir. Those interested should consult it in the original.]

H. B. F.

GONDER (Richard). **Experimentelle Studien mit Trypanosomen und Spironemen (Spirochäten).** [Experimental Studies on Trypanosomes and Spirochaetes.]—*Zeitschr. f. Immunitätsforschung u. Exper. Therapie.* 1912. Nov. 2. Vol. 15. Nos. 2 & 3. pp. 257-291. With 1 plate.

The author's summary of this paper, which is divided into four parts, is to this effect—

(1) Diminution of avidity, in EHRLICH's sense, can be made visible to the eye. Normal trypanosomes stain with certain orthochinoid substances during life; arsenic-fast trypanosomes, on the other hand, stain only after death.

(2) Certain orthochinoid substances cause the blepharoplasts of many trypanosomes to disappear. The chemical attacks the blepharoplast directly; in the case of *Trypanosoma lewisi* this endeavours to reach the macronucleus. Oxidation (LAVERAN and ROUDSKY) seems not to take place. (3) Trypoflavin, arsenophenylglycin, and salvarsan become fixed *in vitro* in the body of the trypanosome, as is shown by animal experiments. Trypanosomes treated *in vitro* with these drugs are not capable of infecting mice. (4) The serum of animals injected intravenously with salvarsan is, a short time after, even *in vitro*, active on *S. recurrentis* and *S. gallinarum*, and also acts therapeutically.

(1) The conclusion is supported by a series of tables. These show that between normal and arsenic-fast nagana strains there is a great difference. Normal trypanosomes placed in contact with oxazine solution stain during life very quickly and then soon die; arsenic-fast trypanosomes do not stain in life and remain alive for a much longer time in the oxazine solution. Whereas a dilution of 1:150,000 oxazine kills the normal trypanosomes in six hours and can stain them after half an hour, the same concentration is entirely without influence on arsenic-fast trypanosomes, even though it acts for a much longer time.

(2) The conclusion drawn from the observations is that a series of orthochinoid substances such as pyronin, oxazine, and trypoflavin, produces a morphological change in the trypanosomes, *viz.* a disappearance of the blepharoplast. In the case of *T. brucei* the destruction of the blepharoplast takes place on the spot owing to the fact that these trypanosomes or their blepharoplasts have a great avidity towards the dye. Other trypanosomes, such as *T. lewisi*, are much more resistant. In the case of *Trypanosoma lewisi* the blepharoplast seeks to reach the macronucleus, but succumbs on the way.

(3) Here again the conclusions are supported by the tables.

A coloured plate shows the action of various substances on the blepharoplasts of trypanosomes.

A. G. B.

Bettencourt (A.) & Borges (I.). **Présence de Trypanosomes dans le Sang des Bovidés portugais.**—*Bull. Soc. Path. Exot.* 1912. Nov. Vol. 5. No. 9. p. 725.

Reference is made to a previous paper (see this *Bulletin*, No. 3, p. 139). Between June and September 62 cattle were examined. Cultural flagellates were found in 25, all from cattle from Portugal. 23 cattle from the Azores gave no flagellates. The authors suggest that this might be due to the absence of the invertebrate transmitter from the Azores.

A. G. B.

Brumpt (E.). **Pénétration du *Schizotrypanum Cruzi* à travers la Muqueuse Oculaire Saine.**—*Bull. Soc. Path. Exot.* 1912. Nov. Vol. 5. No. 9. pp. 723-724.

The author states that *Cercopithecus ruber* is the monkey which is most susceptible to infection by *Schizotrypanum cruzi*, just as it is to infection by *T. gambiense*. One of these monkeys was bitten by *Conorhinus megistus*, the dejecta of which were infective, another by *Cimex lectularius* also with infective dejecta; neither became infected. The author is uncertain whether one should conclude that Conorhinus never transmits the disease by biting or whether it is a question of accidental localisation of the parasite in the salivary glands. *S. cruzi* has a great power of penetration since it can make its way into almost all the cells of the body. The author therefore experimented to see whether the dejecta of Conorhinus can infect by penetrating the healthy

skin or mucous membrane. Experiments in which dejecta were placed on the skin of monkeys and rats were negative. On the other hand, dejecta placed on the ocular globe of an adult *Cercopithecus ruber* infected it, and the animal died as quickly as it would have done had it been inoculated in the peritoneum. In the course of an autopsy on a highly infected rat Brumpt got some virulent blood in his eye; some drops of silver nitrate solution were instilled a few minutes later and, he believes, prevented infection.

A. G. B.

ANNUAL REPORTS FROM BRITISH AFRICAN COLONIES.

COLONY OF THE GAMBIA. **Annual Report of the Medical Department for the Year 1911.** [CHARTRES (E. A.), Senior Medical Officer.]—38 pp. 1912. Printed by Waterlow & Sons, Ltd., London.

Smallpox.—A small outbreak of smallpox occurred during the months of January and April. The infection was traced to labourers who came from French territory. Twelve cases were admitted; none proved fatal. 2,491 successful vaccinations were done in Bathurst and 394 in the Protectorate. The lymph used was supplied from the Incorporated Liverpool Institute of Comparative Pathology and the results continued to be as good as in former years.

Yellow fever.—There were three outbreaks of this disease in May, July and November—four cases occurred in May, with three deaths; six in July, with five deaths; and one fatal case in November. For prevention the ordinary anti-mosquito methods were adopted. Ships were anchored 1,000 yards from the shore, and no direct communication was allowed; all cutters and boats going alongside were examined for mosquitoes and fumigated; labourers and others employed were examined by a Medical Officer, and no cargo or passengers were taken on board. The disease now seems to have died out. [A fuller account appears on p. 250.]

G. C. Low.

SIERRA LEONE. **Annual Report on the Medical Department for the Year ended 31st December, 1911.** [FORDE (R. M.), Principal Medical Officer.]—80 pp. 68 illustrations and 3 charts. 1912. Printed by Waterlow & Sons, Ltd., London.

The general health of the European officials was satisfactory; they numbered 192 during the year (an increase of 21 over the previous year). There was no death from disease. The following climatic diseases were noted amongst them: blackwater fever 1 case; dysentery 1; malaria 27; syriasis 1; liver abscess 1; liver congestion 4.

As regards the general population the following diseases are specially noted.—

Small pox.—There was no epidemic outbreak of this disease during the year. Only 5 cases were reported. 8,432 vaccinations were carried out with 5,199 successes.

Yellow fever.—No case of this disease was met with during 1911.

Dysentery.—197 cases of dysentery [variety not stated] were reported.

Malaria.—2,208 cases were treated during the year, *i.e.*, 264 less than in 1910 for the Colony and Protectorate and 151 fewer cases for Freetown. This points to the efficiency of the mosquito eradication which is now being carried on in Freetown.

Yaws.—This disease does not seem to be progressive in Sierra Leone, much fewer cases (66) having been treated in 1911 than in the previous year.

Beriberi.—A lessened consumption of imported rice has reduced the number of cases of this disease as compared with 1910. Only 8 cases were met with.

Leprosy.—The disease does not seem to be on the increase. Exclusive of cases in the male leprosy segregation ward at Kissy, only 10 were reported.

Trypanosomiasis.—No cases reported.

Ankylostomiasis.—The parasite is common but treatment for conditions resulting from infection is not very frequently sought. During 1911 only 8 cases were treated.

Filariasis.—See this *Bulletin*, No. 2, p. 92.

Quarantine.—During the year it was not found necessary to open the Sanitary Station for the isolation of passengers and crew arriving at the port of Freetown from infected places. The old lazaretto at Kissy, chiefly used for the segregation of small-pox contacts arriving from infected ships or ports, was opened once for the isolation and observation of 45 natives (passengers and crew) who were landed from a steamer on which a case of smallpox was said to have occurred. The case, however, turned out to be one of chicken pox.

An excellent series of photographs illustrates the sanitary part of the report. They show very clearly the difficulties the Sanitary Officer has to contend with in these parts.

G. C. L.

GOVERNMENT OF THE GOLD COAST. **Medical and Sanitary Report for the Year 1911.** [TWEEDY (E. H.), Acting Principal Medical Officer, & RICE (T. E.), Senior Sanitary Officer.]—208 pp. 9 diagrams and charts. 1912. London: Waterlow & Sons, Ltd., Printers.

Small-pox.—This disease was very common during the year in the Eastern Province and although every means was taken to stamp it out by vaccination and isolation the infection was kept alive by the natives adopting the direct inoculation method. Slight outbreaks also occurred in the Central and Western Provinces of the Colony but were easily dealt with. 18,895 successful vaccinations were performed during the year. As regards Ashanti an epidemic started in 1910 and continued for about four months in 1911 when it was stamped out.

Sleeping Sickness.—Cases of this disease were prevalent in the North Western Province of Ashanti, seventeen deaths having been reported. In other parts of Ashanti twelve cases were under treatment with atoxyl, and at the end of a course extending over a period of five or six months the parasites disappeared from the blood and the patients were able to resume their ordinary work.

Blackwater Fever.—Eight patients suffering from blackwater fever were admitted into hospital during the year; four died. Three cases occurred at Accra, two at Seccondee, two at Axim and

one at Tamale. The history of all showed great exposure to infection or carelessness in the mode of living, quinine being taken irregularly or not at all; and in one case a mosquito net was seldom used. Six other cases occurred in Ashanti, three amongst the mining community and three in Syrian petty traders; of this number two ended fatally.

Yellow Fever.—During the year nine cases occurred; seven during May and June, one in February and one in December. It is difficult to account for these latter two. Their occurrence suggests a latent infection, and such isolated outbreaks will only be prevented from becoming general by vigorous action on the part of the Sanitary Staff.

The following measures were taken to prevent the spread of infection at Accra:—

"1. All contacts were isolated and kept under observation for a period of six days.

2. All the non-immunes in the neighbourhood were kept under observation and the temperature of each was taken twice daily.

3. Great care was taken to enquire into all deaths and cases of rise of temperature amongst the natives.

4. The infected house and all buildings in the vicinity were sealed and fumigated with sulphur gas, either by the Clayton machine or by burning the sulphur in open basins. A number of Alformant lamps were distributed amongst the non-immunes living in the native town.

Since no other case occurred, the measures may be stated to have been successful."

The isolated case which occurred on the 21st December, 1911, lived on Railway Hill. After this all the Europeans were removed from that area and kept under observation. All the buildings on the hill were fumigated. No further cases occurred.

In addition to these local measures general works were undertaken to prevent the disease, these including the destruction of mosquitoes and the introduction of pipe-borne water into the different towns.

Plague.—No case of plague was reported during the year. A general campaign has been carried on, however, against rats. All of those examined gave negative results.

G. C. L.

SOUTHERN NIGERIA. **Annual Report on the Medical Department for the Year 1911.** [HOOD (T.), Acting Principal Medical Officer.]—63 + xcv. pp. 1912. Printed by the Government Printer, Lagos.

Except at Lagos and Ebute Metta, vital statistics are unreliable and it is only possible to state that the public health in the Colony and Protectorate has been fairly good during 1911.

In Lagos the incidence of infant mortality is high and deaths connected with pregnancy are of frequent occurrence. The establishment of a Lying-in Hospital is under consideration, in which case it is hoped that principles of infant hygiene will be taught and that midwives will be suitably trained.

Malaria is widespread throughout the whole of Southern Nigeria and it is estimated that quite ninety per cent. of the cases are of the aestivo-autumnal variety.

Blackwater Fever.—26 cases were reported during 1911. There were seventeen recoveries and nine deaths. Eleven officials contracted the disease and seven recovered. Five cases occurred amongst the subordinate European staff of the railway.

Trypanosomiasis.—The disease is probably endemic in the Niger Delta; only five cases however came under observation during the year and two of these were certainly imported from Fernando Po. As soon as cases are discovered they are segregated in fly-proof rooms.

Yellow Fever has not been observed during the year. A case of fever which occurred in a native at Forcados suggested a mild type of this disease; the patient recovered and no other case occurred.

Beriberi.—From time to time patients suffering from beriberi are seen in Lagos. The majority are Kroomen and not natives of the Colony. Kroomen are for the most part engaged on ships or by the merchants as coopers and labourers, and receive rations of rice. They are not accustomed to living on yams, the staple food of the natives of Southern Nigeria. The disease in their case probably results from the consumption of inferior imported rice.

Epidemic Dropsy.—Towards the end of the year a disease, resembling the description given of epidemic dropsy, broke out in the Abeokuta Prison. There were 25 cases and 9 deaths. Whether this outbreak was a form of beriberi or not it is difficult to say, but in none of the patients was there any marked irregularity of the heart, or loss of knee jerks or hyperaesthesia of the calf muscles; and oedema of the legs was a late symptom. The exciting causes were probably overcrowding and unsuitable food, as immediate improvement took place on removing the patients to the sea shore and providing them with a generous meat and fish diet. Prisoners receive a rice ration three times a week, but preparations of yams form the bulk of their diet.

Small-pox.—There was no great epidemic of small-pox in Southern Nigeria. The disease manifested itself mostly at Ibadan and in the neighbourhood of Northern Nigeria. The total number of persons vaccinated during the year was 166,394, being an increase of 30,747 over 1910. Chicken-pox was epidemic at Warri, Onitsha, Calabar, and Ikot-Ekpene.

Ankylostomiasis.—The number of people infected by ankylostomes is extremely high—probably about 75 per cent.—but fortunately the infection seldom gives rise to serious symptoms. Most of the serious cases occurred amongst prisoners, probably because prison life lowers the system.

Dracontiasis.—Patients infected with guinea worm are fairly common in Southern Nigeria. The disease is most prevalent in the Western Province.

Schistosomiasis.—*Schistosomum haematobium* appears to be endemic in the Western Province.

Filariasis.—See this *Bulletin,* No. 2, p. 92.

G. C. L.

NORTHERN NIGERIA. **Annual Medical and Sanitary Report for the Year ending 31st December, 1911.** [MANNING (F.), Acting Principal Medical Officer, & BLAIR (M. Cameron), Senior Sanitary Officer.]—50 pp. 1912. London: Waterlow & Sons, Ltd., Printers.

The average European population during the year was 641, viz., 616 males and 25 females or 368 official and 272 non-official, besides 79 Lagos railway officials resident in Northern Nigeria. As there is now a large mining community with its own medical men, who have not been in the habit of making any returns except in the event of deaths which have to be officially dealt with by the political officers, accurate statistics are difficult to obtain.

Forty European officials were invalided during the year, in twenty-eight instances owing, directly or indirectly, to the effects of climate. Two officials were sent home to undergo the Pasteur treatment, having been bitten by a dog suspected of rabies.

In the sanitary part of the report the tropical diseases which occur in the Protectorate are discussed in detail.

Malaria.—During the year 1,636 cases, not necessarily 1,636 separate persons, were treated for malarial fever—207 Europeans and 1,429 natives, 11 of the latter dying. The figures probably include no native children and very few women; they chiefly refer to adult male employees, African, non-natives, soldiers, police, etc.

At all out-stations where medical officers are posted, regular consignments of kerosene are received for oiling purposes, and grants are given for clearing purposes to out-stations having no prison labour. Clearing of water courses is being carried out at stations as far as is possible with the supply of labour available, and this is well done at the three head-quarter stations in addition to Kano, Zaria, and Minna. Rain water tanks are protected by gauze, and where the gauze is defective are oiled as well. The use of the mosquito net is universal among those Europeans who have not secured mosquito-proof rooms, and is extending among the natives. Most Europeans take a daily five grain dose of quinine.

Blackwater fever.—This disease accounted for the death of six Europeans within the Protectorate.

Trypanosomiasis.—During the year 27 cases of trypanosomiasis were recognised; two of them ended fatally.

The clearing of bush likely to afford cover for tsetse flies is systematically carried out at all stations, and certain high roads are widened where they traverse fly-belts. As sleeping sickness was alleged to be in existence at Jigindi, a town in the Province of Nassarawa, Dr. PARSONS, the medical officer at Keffi, went to investigate. The history of several cases which had ended fatally was very suggestive of the disease, but the parasite could not be observed in the blood of any of the persons examined. Dr. Parsons, however, persuaded the people to move to a new town in a fly-free area.

Yellow fever is unknown in the Protectorate. *Stegomyia calopus* however is not uncommon along the courses of the great rivers;

e.g., it constituted 22 per cent. of the mosquitoes caught at Lokoja during the year; but it is not plentiful at the northern and inland stations, where it is seldom observed during the dry season. All passenger steamers are regularly inspected at Lokoja and at Baro; and this inspection, together with the putting of Burutu Beach entirely under the sanitary authorities of Southern Nigeria, constitutes the first line of defence against invasion by the disease.

Smallpox.—Of 80 cases treated 14 died, all natives. Endemic smallpox, which assumes an epidemic form annually, is very prevalent throughout the Protectorate, but at present it is impossible to compile accurate statistics of it. It generally begins to show itself in the second half of the dry season and, as a rule, dies down when the rainy season has seriously set in.

During the year 5,494 vaccinations were performed, and 3,879 proved successful. The fair number of successes recorded in the dry hot interior of the country continues to demonstrate the efficacy of powdered lymph. Constant efforts are directed towards the breaking down of the prejudice against vaccination which is harboured by many natives.

Dysentery.—During the year 26 European and 603 native cases were treated; 37 of the latter proved fatal [variety of disease not stated]. All possible steps are taken to induce the natives to safeguard their water-supplies by applying parapets to the mouths of their wells, by digging new wells in suitable places, and by closing wells which are known to be badly contaminated.

Leprosy.—84 cases were treated. The number of lepers is very large, but cannot be accurately determined.

Schistosomiasis.—6 cases of *S. haematobium* were treated.

Dracontiasis.—82 cases appear in the returns. The disease is common, possibly as common as dysentery in many places.

Filariasis.—See this *Bulletin* No. 2, p. 91.

General measures, such as sewage disposal, disposal of refuse, water supplies, drainage and clearance of bush, undergrowth, etc., are also dealt with in this interesting report.

G. C. L.

UGANDA. **Extracts from the Annual Medical Report for the Year ended 31st December, 1911, by the Principal Medical Officer, Uganda Protectorate.** [HODGES (A. D. P.)]—Unpublished Report to the Secretary of State for the Colonies.

Malaria was generally reported to have been more prevalent during the latter part of, and immediately after, the rainy seasons, that is, from May to July and from November to mid-January. The general type was mild and there was no exceptional increase or decrease in prevalence during the year. The percentage of natives treated was slightly higher and that of Europeans slightly lower than in the previous year. The great majority of the cases were aestivo-autumnal or tropical malaria, which continues to be the most important cause of sickness among Europeans and Asiatics in Uganda. The total number of cases treated was 5,286, including 203 Europeans. The total in 1910 was 4,078, including 225 Europeans.

Blackwater fever.—So far as seasonal prevalence is shown, it corresponds with that for malaria. The number of cases was considerably less, with a less mortality than in 1910, 18 cases with 3 deaths occurring as compared with 26 cases and 6 deaths.

Dengue was less prevalent, 69 cases being reported of which only one was admitted to hospital.

Yellow fever is as yet unknown in Uganda, though the carrier is common.

Elephantiasis.—Thirteen cases were reported as compared with eleven the previous year. Only one was admitted to hospital. This disease is more prevalent in the Eastern and Northern Provinces than elsewhere, and in certain localities is believed to be fairly common, but it seldom comes under medical observation.

Trypanosomiasis.—See this *Bulletin* No. 3. p. 135.

Relapsing or *spirillum fever* showed no sign of seasonal variation. It was much less prevalent among Europeans than among natives. The carrier, *Ornithodorus moubata,* is not yet known to exist in the Northern (Nile) Province and is comparatively rare in the Eastern (Busoga) Province, where it is of recent introduction, but in the rest of the Protectorate it infests native dwellings and traffic routes.

Dysentery was less prevalent than in 1910, and no serious epidemic was reported. The disease is more frequent in the Western Province (Ankole and Toro) than elsewhere, and is, on the whole, more common in the dry season.

Leprosy is not common, though it occurs throughout the greater part of the Protectorate. Hitherto Medical Officers have not come in contact with it much, but cases are likely to increase in the future as hospitals and dispensary accommodation is extended, and the confidence of the natives increases. Twenty-two cases were returned in 1911, sixteen in 1910 and eight in 1909; twelve of the twenty-two were of the nodular and ten of the anaesthetic type.

Undulant fever.—Twenty cases were returned. The disease is endemic in the Western Province and the extreme south of Buganda. In the former district it has been confused, under the name *muhinyo,* with sleeping sickness and other diseases, including probably beriberi. Large numbers of deaths are attributed to *muhinyo* in the native returns.

Plague.—" The return of cases which came under treatment is 34 with 29 deaths. There were 17 with 13 deaths in 1910, and 74 with 37 deaths in 1909. All the cases were admitted at Mbale (Bukedi) except one case, which was admitted at Jinja and which was infected at, or en route from Mbale. The deaths reported in native returns from the Bukedi districts for the last three years are as under:—

1909	 682	deaths
1910	 1,659	,,
1911	 1,773	,,

Although these returns cannot be regarded as complete, they are improving in accuracy," and they tend to show that plague is on

the increase in that district. "The greatest prevalence, as shown by native returns, was during the period from June to September and the least from October to December. All forms of the disease were seen, namely, bubonic, pneumonic, septicaemic and pestis minor. The type was, on the whole, severe and the mortality high. As has been previously reported, plague is widely endemic in the Protectorate, and minor epidemics, which are seldom heard of, occur from time to time among the natives of various parts. It has long been known to be endemic in the south-eastern part of Buganda, bordering on German East Africa, but no serious epidemic has occurred there during recent years." From time to time natives have reported large numbers of deaths in Busoga and other parts of the country but on investigation these have not been found to be due to plague. All rapidly fatal illnesses are generally attributed to that disease by the natives.

In the part of the report dealing with sanitation it is stated that the chiefs and others have been instructed to report all cases and all deaths from plague as they occur. Native inspectors are employed to see that this instruction is carried out and also instructions with regard to isolation of infected persons and contacts and the burning or disinfection of clothing and huts.

A temporary plague hospital with an isolation ward for contacts exists at Mbale, the chief station in the endemic area. The chiefs are encouraged to build segregation huts in the villages where the disease is endemic, and to place the sick in these as they are known. The Medical Officer reports that this is being largely done. Huts in which plague has occurred were burnt when possible and the rats destroyed. Compensation was given for huts and clothes which were burnt.

Haffkine's serum was used during the year and 197 persons vaccinated.

Plague has not yet occurred among the Asiatics, who number about 200 in Bukedi. This may be partly due to the fact that they all keep cats.

General measures are also given for sewage disposal, the disposal of refuse, clearance of bush and undergrowth, etc. A circular concerning leprosy has been issued to Medical Officers during the year and recommendations for future work are given in detail.

G. C. L.

NYASALAND PROTECTORATE. **Annual Report on the Medical Department for the Year ended 31st March, 1912.** [HEARSEY (H.), Principal Medical Officer.]—56 pp. Zomba: Printed by the Government Printer.

Malaria.—The seasonal incidence of malaria corresponded with that of previous years, the largest proportion of cases occurring during and shortly after the wet season. It should be remembered, however, that Europeans may become infected whilst travelling and this circumstance needs to be borne in mind when considering the seasonal distribution of cases among this com-

munity. There were 808 cases of malaria treated in 1911-1912, as compared with 786 in the year preceding.

Blackwater Fever.—Five cases of blackwater fever, as against three in the previous year, came under the care of the Medical Officers, this number including one Asiatic. Two of these cases had a fatal termination, namely, one European and the Asiatic referred to. It is noteworthy that notwithstanding the appreciable increase in the European population within recent years, the number of cases of blackwater fever has perceptibly diminished. This no doubt is mainly to be attributed to the greater attention which is now devoted to personal prophylaxis as regards malaria.

Tick Fever.—This disease is prevalent in the districts bordering the Lake but rarely comes under the notice of the Medical Staff.

Filariasis.—A few cases of elephantiasis occurred, mostly in the Lower Shire and North Nyasa districts. The scrotum would appear to be involved almost as frequently as the lower extremities.

Smallpox.—Seventy-eight cases of this disease, with fifteen deaths, were recorded during the year, as compared with 236 in the previous year. Notwithstanding that these cases were reported from six widely separated localities, in consequence of the promptness of the preventive measures adopted there was no spread of the infection to neighbouring villages. In two of the areas the infection was clearly demonstrated to have been introduced by natives entering the Protectorate from adjacent territories, and this is a danger which needs to be constantly guarded against.

Dysentery.—There were 317 cases of dysentery, with five deaths. The disease reaches its maximum incidence at the beginning of the wet season, owing to the scarcity, and presumably the pollution, of the then existing water supplies.

Beriberi.—There were eleven cases of beriberi, with one death, as against 24 in 1910.

Pellagra.—In the year 1910-11, Stannus observed a number of cases of a skin eruption among the inmates of the Central Prison at Zomba, which he diagnosed as pellagra.

Leprosy.—Only four cases of leprosy appear in the returns. Twenty cases however were observed in the Lower Shire district (population 24,940), and 33 in the Ruo district (population 26,680). The disease has a higher incidence in the neighbourhood of Lake Shirwa in the Zomba district, and a comparatively large number of cases are reported to exist in the Upper Shire district.

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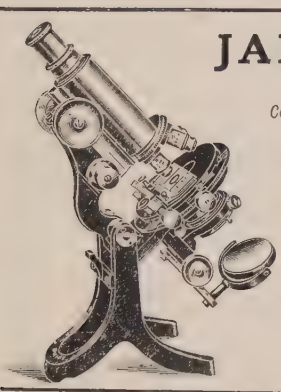

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